
Dr. Carol Rivers'
PREPARING FOR THE WRITTEN BOARD EXAM
IN EMERGENCY MEDICINE

Seventh Edition
Volume II

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The object of education is not learning, but discipline
and enlightenment of the mind.

Woodrow Wilson

Dr. Carol Rivers' Preparing for the Written Board Exam in Emergency Medicine, Seventh Edition

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TABLE OF CONTENTS

VOLUME I

<i>Foreword</i>	xii
<i>Introduction</i>	xiii
<i>Recommended Study Plan</i>	xv
Cardiovascular Disorders	1
Head, Ear, Eye, Nose, and Throat Disorders	121
Abdominal and Gastrointestinal Disorders	215
Thoracic and Respiratory Disorders	299
Traumatic Disorders	377
Orthopedic Emergencies	431
Musculoskeletal Disorders (Nontraumatic)	495
Nervous System Disorders	515
Gynecologic and Obstetric Disorders	557

VOLUME II

Pediatric Emergencies	615
Toxicologic Disorders	689
Endocrine, Metabolic, and Nutritional Disorders	747
Environmental Disorders	805
Psychobehavioral Disorders	857
Hematologic Disorders	907
Oncologic Disorders	943
Systemic Infectious Disorders	959
Immune System Disorders	989
Renal and Urologic Disorders	1013
Cutaneous Disorders	1051
Emergency Medical Services	1093
Procedures and Skills Integral to the Practice of Emergency Medicine	1111
Other Core Competencies of the Practice of Emergency Medicine	1169
Emergency Department Administration	1175
Ethical-Legal Aspects of Emergency Medicine	1181
Physician-Patient Interaction	1200
Professionalism	1208
<i>Mechanics of the Written Board Exam</i>	1217
<i>Additional Tips for Good Performance</i>	1227

PEDIATRIC EMERGENCIES

Life-Threatening Cardiac Dysrhythmias	625
Cardiopulmonary Resuscitation	625
Airway	625
Breathing	629
Circulation	629
Chest Compressions	629
Defibrillation and Cardioversion	630
Vascular Access	631
Life-Threatening Cardiac Arrhythmias	632
Unique Issues in Neonates and Infants	634
Risk Factors Associated with Neonatal Cardiopulmonary Arrest	634
APGAR Scoring	635
Meconium Staining	636
Neonatal Seizures	636
Congenital Diaphragmatic Hernia	637
Tracheoesophageal Fistula	637
Omphalocele and Gastroschisis	637
Necrotizing Enterocolitis	638
Cyanosis	639
Congestive Heart Failure	639
Jaundice in the Newborn	640
Apparent Life-Threatening Event	641
Sudden Infant Death Syndrome	642
Congenital Heart Disease	643
Cyanotic Heart Lesions	643
Acyanotic Heart Lesions	644
Ductal Dependent Lesions	644
Congestive Heart Failure	645
Airway Emergencies	646
Upper Airway	646
Lower Airway	649
Pediatric Infectious Diseases	655
The Febrile Child	655
Bacteremia and Sepsis	657
Meningitis	658
Pneumonia	660
Pertussis (Whooping Cough)	663

Otitis Media	664
Urinary Tract Infection	666
Kawasaki Disease (Mucocutaneous Lymph Node Syndrome)	668
Pediatric Seizures.....	670
Cerebrospinal Fluid Shunts	673
Pediatric Gastrointestinal/Genitourinary Emergencies.....	676
Child Abuse.....	681
Child Neglect	681
Munchausen Syndrome by Proxy (Polle Syndrome)	681
Sexual Abuse	682
Physical Abuse.....	683

PEDIATRIC EMERGENCIES: SELF-ASSESSMENT QUESTIONS

1. The appropriate endotracheal tube for a 6-year-old is:
 - (a) 4.5 uncuffed
 - (b) 4.5 cuffed
 - (c) 5.5 uncuffed
 - (d) 5.5 cuffed
2. Relative to the location of the adult airway in the neck, the child's airway is:
 - (a) More anterior and higher
 - (b) More posterior and higher
 - (c) More posterior and lower
 - (d) More anterior and lower
3. Which of the following drugs should not be administered via the endotracheal route?
 - (a) Atropine
 - (b) Calcium chloride
 - (c) Epinephrine
 - (d) Naloxone
4. Which of the following is not considered a risk factor for sudden infant death syndrome?
 - (a) Maternal age >35 years old
 - (b) Low birth weight
 - (c) Smoking and drug abuse by mother
 - (d) None of the above
5. All of the following statements regarding congenital diaphragmatic hernias are accurate except:
 - (a) Left-sided hernias are more common than right-sided hernias.
 - (b) Associated polyhydramnios is common.
 - (c) Treatment is intubation, orogastric tube placement, IV hydration, and surgical repair.
 - (d) Right-sided hernias are called Bochdalek hernias.
6. Which of the following is not a structural component of the tetralogy of Fallot?
 - (a) Pulmonic stenosis
 - (b) Ventricular septal defect
 - (c) Left ventricular hypertrophy
 - (d) Dextroposition and overriding of the aorta

7. Initial management of a hypercyanotic ("Tet") spell in a child with tetralogy of Fallot should include all of the following except:
- (a) Administration of supplemental oxygen
 - (b) Placement of the child in the prone knee-chest position
 - (c) Morphine 0.1 mg/kg IV, IM, or SC
 - (d) Propranolol 0.05–0.1 mg/kg IV
8. Which of the lesions listed below is an acyanotic heart lesion?
- (a) Transposition of the great vessels
 - (b) Aortic stenosis
 - (c) Total anomalous pulmonary venous return
 - (d) Tricuspid atresia
9. Which of the following statements regarding necrotizing enterocolitis is least accurate?
- (a) Premature infants with very low birth weight are most commonly affected.
 - (b) It typically develops during the first 2 weeks of life.
 - (c) The first and most frequent clinical finding is abdominal wall erythema.
 - (d) Pneumatosis intestinalis is the radiographic hallmark.
10. The presence of biphasic stridor on examination localizes the airway obstruction _____.
- (a) Above the larynx
 - (b) At the larynx
 - (c) Below the larynx
 - (d) Below the carina
11. The usual causative organism of bronchiolitis is:
- (a) Parainfluenza virus
 - (b) Respiratory syncytial virus
 - (c) Adenovirus
 - (d) Influenza B virus
12. The usual causative organism of croup is:
- (a) Respiratory syncytial virus
 - (b) Parainfluenza virus
 - (c) Adenovirus
 - (d) Echovirus

13. Anticholinergic agents (atropine, ipratropium bromide) produce their beneficial effects in asthma by:
- (a) Increasing cyclic GMP
 - (b) Decreasing cyclic GMP
 - (c) Increasing cyclic AMP
 - (d) Decreasing cyclic AMP
14. All of the following poisonings may be associated with hyperpyrexia except:
- (a) Atropine
 - (b) Salicylates
 - (c) Amphetamines
 - (d) Acetaminophen
15. Under normal land conditions, the greatest amount of heat loss from the body occurs via:
- (a) Radiation
 - (b) Evaporation
 - (c) Convection
 - (d) Conduction
16. The most common bacterial causative organism of otitis media is:
- (a) *Streptococcus pneumoniae*
 - (b) *Haemophilus influenzae* nontypable
 - (c) *Moraxella catarrhalis*
 - (d) *Staphylococcus aureus*
17. The pathogens most often responsible for the production of pneumonia in children ≥ 5 years old are:
- (a) *Haemophilus influenzae* and *Streptococcus pneumoniae*
 - (b) *Haemophilus influenzae* and *Mycoplasma pneumoniae*
 - (c) *Mycoplasma pneumoniae* and *Streptococcus pneumoniae*
 - (d) *Mycoplasma pneumoniae* and viruses
18. A 3-week-old infant is brought in for evaluation of a staccato cough. His mother states that he was born by normal spontaneous vaginal delivery and that, aside from some nasal congestion and eye inflammation over the past few days, he had been well until yesterday. On examination, the child is afebrile but quite tachypneic and has bilateral conjunctivitis. Pulse oximetry reveals a saturation of 94%, and chest radiograph reveals bilateral interstitial infiltrates. The organism most likely to produce this constellation of signs and symptoms in an infant this age is:
- (a) Group B streptococci
 - (b) *Chlamydia trachomatis*
 - (c) *Staphylococcus aureus*
 - (d) *Mycoplasma pneumoniae*

19. The most common bacterial pathogen in all age groups beyond the newborn period is:
- (a) *Streptococcus pneumoniae*
 - (b) *Haemophilus influenzae* type B
 - (c) *Staphylococcus aureus*
 - (d) Group B streptococci
20. When evaluating a child who presents with pneumonia, findings on the CBC (although certainly not diagnostic) can suggest a particular etiologic agent. The classic WBC findings that occur in association with particular pathogens are listed below. Which of the following findings is inappropriately matched?
- (a) Normal WBC count and differential/*Mycoplasma pneumoniae*
 - (b) High WBC count with a left shift/*Streptococcus pneumoniae*
 - (c) Marked lymphocytosis/viruses
 - (d) Eosinophilia/*Chlamydia trachomatis*
21. In children, cardiac arrest is most commonly secondary to:
- (a) A primary cardiac event
 - (b) Respiratory arrest
 - (c) Shock
 - (d) None of the above
22. The easiest and most accurate method used in the selection of an endotracheal tube for pediatric intubation is:
- (a) A formula that estimates the tube size based on the child's age
 - (b) A tube size that equals the diameter of the tip of the child's little finger
 - (c) A tube size that approximates the size of the child's nostril
 - (d) A length-based resuscitation tape
23. You are attempting to defibrillate a child weighing 20 kg. What dose should be used for the initial attempt?
- (a) 20 joules
 - (b) 40 joules
 - (c) 60 joules
 - (d) 80 joules
24. When using the endotracheal route to administer epinephrine during pediatric resuscitation, the concentration of epinephrine should be _____ that administered when the IV route is used.
- (a) The same as
 - (b) Half
 - (c) Double
 - (d) Ten times

25. You have just delivered a baby in a rural emergency department. At 1 minute, the infant is limp, has a slow and irregular respiratory effort, a heart rate of 80 beats per minute, acrocyanosis, and some reflex irritability. The appropriate 1-minute APGAR score for this infant is:
- (a) 2
 - (b) 3
 - (c) 4
 - (d) 5
26. You are evaluating a 2-year-old child for possible retropharyngeal abscess and decide to obtain a soft-tissue lateral radiograph of the neck. For an accurate interpretation, the radiograph must be taken:
- (a) During inspiration with the neck in slight extension
 - (b) During inspiration with the neck in slight flexion
 - (c) During expiration with the neck in slight extension
 - (d) During expiration with the neck in slight flexion
27. Which of the following statements regarding bronchiolitis is inaccurate?
- (a) It usually affects infants 2 months to 2 years old.
 - (b) The causative organism is generally respiratory syncytial virus.
 - (c) Apnea is a serious complication, and premature infants <6 months old are at greatest risk.
 - (d) Aerosolized ribavirin is indicated for all infants hospitalized with this illness.
28. Which statement below regarding occult bacteremia is inaccurate?
- (a) Children 3–36 months old are most commonly affected.
 - (b) *Streptococcus pneumoniae* is responsible for most cases.
 - (c) The risk of occult bacteremia is increased in the presence of a WBC count $\geq 15,000/\text{mm}^3$ and a temperature $\geq 102.2^\circ\text{F}$ (39°C).
 - (d) The risk of progression to a major focus of infection, eg, meningitis, is the same for all infecting agents.
29. The most important complications of Kawasaki disease are:
- (a) Hematologic
 - (b) Gastrointestinal
 - (c) Cardiovascular
 - (d) Neurologic
30. Which of the following is not one of the criteria used to diagnose Kawasaki disease?
- (a) Fever ≥ 5 days
 - (b) ECG changes
 - (c) Polymorphous rash
 - (d) Bilateral nonpurpurative conjunctivitis

31. The most appropriate management for a young child with Kawasaki disease is:
- (a) Admission for IV gamma globulin and high-dose aspirin therapy
 - (b) Admission for IV antibiotics and steroids
 - (c) Discharge on high-dose aspirin therapy with follow-up in 2–3 days
 - (d) Discharge on high-dose aspirin therapy and an oral second-generation cephalosporin
32. All of the following are characteristic of simple febrile seizures except:
- (a) Duration <15 minutes
 - (b) Generalized convulsion
 - (c) Multiple episodes in a 24-hour period
 - (d) Absence of focal postictal neurologic deficits
33. The agent of choice for rapid-sequence intubation of an asthmatic patient in respiratory failure is:
- (a) Succinylcholine
 - (b) Midazolam
 - (c) Ketamine
 - (d) Pancuronium
34. Which of the following statements regarding an omphalocele and its management is least accurate?
- (a) It is a defect in the umbilical ring that results in protrusion of the intestines outside the abdominal wall.
 - (b) The protruding intestines are not covered by a peritoneal sac.
 - (c) Associated anomalies are seen in one-third to one-half of these infants.
 - (d) Management consists of placing an orogastric tube, covering the eviscerated intestines with saline-soaked sterile gauze and placing them in a plastic bag, administering IV fluids along with prophylactic antibiotics, and obtaining immediate pediatric surgery consult.
35. What is the most common cause of congestive heart failure in infants and young children?
- (a) Congenital heart disease
 - (b) Dysrhythmias (supraventricular tachycardia, ventricular tachycardia)
 - (c) Kawasaki disease
 - (d) Severe anemia
36. The drug of choice for the treatment of supraventricular tachycardia in children is:
- (a) Adenosine
 - (b) Propranolol
 - (c) Verapamil
 - (d) Procainamide

37. Which of the following statements regarding prostaglandin E_1 is inaccurate:
- (a) It is a potent dilator of the ductus arteriosus.
 - (b) It can be used to maintain the patency of (or reopen) the ductus arteriosus in infants with ductal-dependent congenital heart lesions.
 - (c) Significant adverse effects associated with its use include apnea and hypotension.
 - (d) The initial infusion rate is 1 mcg/kg/min.
38. The organism(s) most commonly responsible for causing CSF shunt infections are:
- (a) *Staphylococcus* spp
 - (b) *Klebsiella*
 - (c) Gram-negative organisms
 - (d) Group A streptococci
39. The sign/symptom most commonly present in association with CSF shunt infection is:
- (a) Headache
 - (b) Lethargy
 - (c) Fever
 - (d) Meningismus
40. A 10-day-old infant is brought in for evaluation of respiratory distress. His mother states that he was born by normal spontaneous vaginal delivery and that, aside from some nasal congestion and coughing over the past few days, he had been well until yesterday. On examination, the child is afebrile but quite tachypneic with moderate retractions. His pulse is 80 beats per minute, and pulse oximetry is 89% on 100% oxygen. What is the next step in management of this patient?
- (a) Chest radiograph
 - (b) Positive-pressure ventilation
 - (c) Begin chest compressions
 - (d) Epinephrine 0.01 mg/kg IV
41. A 14-day-old infant is brought in for evaluation of respiratory distress. His mother states that he was born by normal spontaneous vaginal delivery but has been having difficulty feeding over the past 24 hours. On examination, the child is afebrile but tachypneic with moderate retractions and cyanotic with pulse oximetry of 80%. After placing him on 100% oxygen, an arterial blood gas is obtained and the PaO_2 is 160 mmHg. Which of the following is least likely?
- (a) Sepsis
 - (b) Congenital heart disease with right to left shunt
 - (c) Pneumonia
 - (d) Primary seizure disorder

ANSWERS

- | | | | | | |
|------|-------|-------|-------|-------|-------|
| 1. d | 8. b | 15. a | 22. d | 29. c | 36. a |
| 2. a | 9. c | 16. a | 23. b | 30. b | 37. d |
| 3. b | 10. c | 17. d | 24. d | 31. a | 38. a |
| 4. a | 11. b | 18. b | 25. c | 32. c | 39. c |
| 5. d | 12. b | 19. a | 26. a | 33. c | 40. b |
| 6. c | 13. b | 20. c | 27. d | 34. b | 41. b |
| 7. d | 14. d | 21. b | 28. d | 35. a | |

Use the pre-chapter multiple choice question worksheet (page xx) to record and determine the percentage of correct answers for this chapter.

I. LIFE-THREATENING CARDIAC DYSRHYTHMIAS

- A. Unlike in adults, in whom rhythm disturbances are primary cardiac disturbances, in children rhythm disturbances are frequently secondary problems.
1. Children at risk of dysrhythmias may have congenital or acquired heart disease, systemic disease, intoxication, or acute hemodynamic alteration.
 2. Cardiac arrest is usually secondary to respiratory arrest. Circulatory failure (shock) is the second most common precipitating event.
 3. Symptomatic sinus bradycardia is generally the result of prolonged hypoxemia or increased vagal tone.
 4. Primary cardiac events (eg, ventricular fibrillation) are rare.
- B. Dysrhythmias by age

Table 25: Most Common Clinically Significant Dysrhythmias by Age

Age (years)	Dysrhythmias
<1	Bradycardia, atrial fibrillation, ventricular fibrillation
1–5	Supraventricular tachycardia, bradycardia, atrial flutter
6–12	Nonspecific dysrhythmia, supraventricular tachycardia, atrial flutter
13–18	Nonspecific dysrhythmia, atrial fibrillation

II. CARDIOPULMONARY RESUSCITATION

A. Airway

1. Foreign body aspiration with complete obstruction
 - a. Infants <1 year old
 - (1) Deliver back blows and chest thrusts.
 - (2) Do not perform the Heimlich maneuver (abdominal thrusts); it may result in laceration of the liver in this age group.
 - (3) If the infant loses consciousness, open the airway with a tongue-jaw lift and inspect the mouth.
 - (4) If a foreign body is seen, remove it; do not perform blind finger sweeps, because they may push the foreign body deeper into the airway.
 - (5) Attempt ventilation.
 - b. Older children
 - (1) Use the Heimlich maneuver.
 - (2) If the child loses consciousness, open the airway with a tongue-jaw lift and inspect the mouth.
 - (3) If a foreign body is seen, remove it; do not perform blind finger sweeps.
 - (4) Attempt ventilation.

2. Intubation

- a. Although intubation in children does not differ significantly from that in adults, anatomic differences in the airway affect the equipment used, the preferred approach, and the potential for complications:
 - (1) Because children have a larger occiput, a towel roll under the shoulders may help align the airway for easier visualization. The larynx is higher and more anterior in infants and toddlers, making the angle for intubation more acute. A straight (Miller) laryngoscope blade is more useful in creating a visual plane from the mouth to the glottis in children <2 years old and is also more effective in controlling the tongue, which is relatively larger in size.
 - (2) The cricoid ring is the narrowest portion of the airway and forms an adequate seal; therefore, uncuffed tubes were traditionally used in children <8 years old. With the introduction of smaller-profile, lower-pressure cuffed tubes, cuffed tubes may be considered in children beyond the newborn period if the cuff pressure can be maintained <20 cm H₂O.
 - (3) The mass of adenoidal tissue is greater and the nasopharyngeal angle is more acute in young children, making nasopharyngeal intubation more difficult and more likely to result in damage to the nasopharyngeal soft tissues; therefore, orotracheal intubation under direct visualization is the preferred method of establishing initial airway control in a child.
 - (4) The trachea is shorter (5 cm at birth); this increases the potential for accidental intubation of the right mainstem bronchus.
- b. General approach
 - (1) Determine the proper tube size.
 - (a) Use a *length*-based resuscitation tape (eg, Broselow® tape). This is the most accurate predictor of correct tracheal tube size and is also useful in determining drug dosages. Tracheal tubes 0.5 mm smaller or larger than the estimated size should be easily available.
 - (b) **If a tape is unavailable, use the following formulas in children >2 years old:**
 - i. **If using an uncuffed endotracheal tube: tracheal tube size = (age [in years]/4) + 4**
 - ii. **If using a cuffed endotracheal tube: tracheal tube size = (age [in years]/4) + 3.5**
 - (c) The appropriate size tracheal tube for premature infants is 2.5–3.0 mm; for newborns 3.0–3.5 mm; for 1-year-olds 4.0–4.5 mm, and for 4-year-olds 5.0 mm.
 - (2) Prepare to intubate.
 - (a) Place the head in the sniffing position.
 - (b) Use the chin-lift or jaw-thrust to move the hypotonic mandibular tissue out of the way.
 - (c) If the respiratory effort is inadequate, preoxygenate with 100% oxygen for 5 minutes (if possible).
 - (d) Apply cricoid pressure (Sellick maneuver) to decrease the likelihood of passive gastric regurgitation and aspiration in unresponsive patients during bag-mask ventilation. Do not release cricoid pressure until the tube is in place. There is insufficient evidence to recommend routine cricoid pressure to prevent aspiration during endotracheal intubation in children. Cricoid pressure should be stopped if it interferes with ventilation or the ease of ventilation.

- (e) Using a straight blade, advance the tip until it is just under the epiglottis. Visualize the larynx by exerting gentle traction upward and away from the intubator (in the direction of the handle of the laryngoscope). The tracheal tube should then be guided through the vocal cords under direct visualization with the black glottic marker placed at the vocal cord level.
- (3) Rapid-sequence intubation
 - (a) Definition: simultaneous administration of potent sedative agent and rapid-acting paralytic drug as well as medications to ameliorate potential adverse effects of laryngoscopy to facilitate endotracheal intubation safely and effectively; safe and effective in children when used properly
 - (b) Medications
 - i. Pretreatment (controversial)
 - Atropine 0.02 mg/kg IV push (minimum 0.1 mg, maximum 0.5 mg): blunts vagal stimulation and prevents bradycardia associated with laryngoscopy.
 - Lidocaine 1–1.5 mg/kg IV push: theoretically blunts increase in intracranial pressure associated with intubation
 - ii. Induction
 - Midazolam 0.1 mg/kg IV push: variable dosing
 - Ketamine 1–2 mg/kg IV push: although previously believed to increase intracranial pressure, newer data suggest this may not be the case.
 - Thiopental 3–5 mg/kg IV push: may cause hypotension, laryngospasm
 - Etomidate 0.3 mg/kg IV push: hemodynamically stable; caution in septic shock secondary to adrenal suppression
 - Propofol 1–2 mg/kg IV push: may cause hypotension, may be used in some institutions
 - iii. Paralytic agents
 - Rocuronium 0.6–1 mg/kg IV push
 - Succinylcholine 1–1.5 mg/kg IV push, infants may require 1.5–2 mg/kg; Contraindications include children with underlying muscular disorders or extensive burns.
- (4) Confirm tracheal tube placement after intubation by:
 - (a) Observing the chest for symmetric movement
 - (b) Listening over the axillae to confirm equal breath sounds
 - (c) Listening over the stomach to assure breath sounds are absent
 - (d) Obtaining a chest radiograph; the tip of the tracheal tube should be located above the carina at the level of T2.
 - (e) End-tidal CO₂ detection
 - i. In patients with a pulse, this is the best adjuvant method (most sensitive/specific) for verification of tube placement; however, the most accurate means is observation of the tube passing through the vocal cords.
 - ii. A false-negative reading can occur with severe circulatory collapse; therefore, if the CO₂ is not detected during CPR, take another look with a laryngoscope.

- (f) Clinical deterioration in a previously stable intubated patient receiving positive-pressure ventilation can be remembered by the mnemonic "DOPE":
- D**islodged tube
 - O**bstructed tube
 - T**ension **P**neumothorax
 - E**quipment failure
- (5) Rescue airway devices: supraglottic devices, eg, the laryngeal mask airway, may quickly be inserted blindly in skilled hands.

Table 26: Laryngeal-Mask Airway Sizes

Age (weight)	Size
Newborn (0–5 kg)	1
3 months to 1 year old (5–10 kg)	1.5
1–4 years old (10–20 kg)	2
4–8 years old (20–30 kg)	2.5
>8 years old (30–40 kg)	3
Adolescent and adult	4

3. Surgical airways in children

a. Needle cricothyrotomy with jet ventilation

- (1) Indications: inability to ventilate and inability to intubate in children <8 years old
- (2) Only a temporary measure
 - (a) Effective for approximately 30–45 minutes
 - (b) Oxygenation is usually adequate, but ventilation is ineffective, resulting in progressive hypercarbia.
- (3) Procedure
 - (a) Position patient in supine position with neck hyperextended.
 - (b) Prepare the neck with antibacterial solution.
 - (c) Identify cricothyroid membrane just below the thyroid cartilage.
 - (d) Using a 12- or 14-gauge over-the-needle catheter attached to a syringe (can be filled with saline), puncture skin in the midline directly over the cricothyroid membrane while stabilizing the trachea with the free hand.
 - (e) Apply negative pressure to the syringe and advance caudally and posteriorly at a 45° angle until air is aspirated (confirms placement in trachea).
 - (f) Once air has been aspirated, advance the catheter into the trachea while withdrawing the needle.
 - (g) Attach the adapter from a 3-mm endotracheal tube to the catheter.
 - (h) Use a jet ventilation device capable of delivering at least 50 psi.

b. Surgical cricothyrotomy may be used in children >8 years old as an alternative to needle cricothyrotomy.

B. Breathing

1. Each breath should be delivered slowly (over 1–1.5 seconds).
2. The appropriate volume of each breath is the volume that causes the chest to rise; this corresponds to a tidal volume of 6–8 mL/kg with spontaneous breathing, 10–15 mL/kg with volume ventilation.
3. Rate (breaths per minute)
 - a. Neonates: 40–60
 - b. Infants <1 year old: 24–40
 - c. Children 1–3 years old: 22–30
 - d. Children >8 years old: 12
4. Mechanical ventilation
 - a. For children, use a volume-limited ventilator. Initial settings:
 - (1) Tidal volume 10–15 mL/kg
 - (2) FiO₂ 100%
 - (3) Inspiratory time 0.5–1.5 seconds
 - (4) Age-appropriate respiratory rate (adjusted to physiologic needs)
 - (a) 20–30 breaths per minute in infants
 - (b) 16–20 breaths per minute in children
 - (5) Positive end-expiratory pressure 3–5 cm H₂O
 - b. For infants <10 kg, use a pressure-limited ventilator with the following initial settings:
 - (1) Peak inspiratory pressure 20–30 cm H₂O; use the lowest level that results in normal chest expansion and good breath sounds.
 - (2) Inspiratory time 0.5–1.0 seconds
 - c. Obtain arterial blood gases 10–15 minutes after start of mechanical ventilation, and adjust settings as needed.
5. Hyperventilation should be reserved for patients with signs of cerebral herniation or suspected pulmonary hypertension.

C. Circulation

1. Normal heart rate (beats per minute) based on age
 - a. Neonate: 100–160
 - b. 1–12 months old: 100–180
 - c. 1–2 years old: 90–150
 - d. 2–4 years old: 75–130
 - e. 4–8 years old: 60–120
 - f. >8 years old: 60–100
2. Normal systolic blood pressure based on age (lowest 5th percentile)
 - a. <1 month old: >60 mmHg
 - b. 1 month to 1 year old: 70 mmHg
 - c. >1 year old: 70 + 2 (age in years) mmHg (estimate)

D. Chest compressions

1. ~~Should be started in:~~
 - a. **Children who do not have a pulse or have a heart rate <60 beats per minute and signs of poor perfusion despite ventilation with 100% oxygen**

b. Neonates with a heart rate <100 beats per minute despite adequate ventilation with 100% oxygen for 30 seconds

2. Compression depth should be at least $\frac{1}{3}$ the total depth of the chest.
3. Technique
 - a. Newborns
 - (1) Rate = 120 events per minute (90 compressions and 30 breaths)
 - (2) Technique: 2 fingers or thumbs with hands encircling chest 1 fingerbreadth below intramammary line
 - (3) Compression/ventilation ratio 3:1 (two-rescuer CPR)
 - (4) Depth = at least $\frac{1}{3}$ the total depth of the chest
 - b. Infants <1 year old
 - (1) Rate = 100 per minute
 - (2) Technique: 2 fingers or thumbs with hands encircling chest 1 fingerbreadth below intramammary line
 - (3) Compression/ventilation ratio 15:2 (two-rescuer CPR)
 - (4) Depth = at least $\frac{1}{3}$ the total depth of the chest or 1.5 inches (4 cm) in most infants
 - c. Children 1–8 years old
 - (1) Rate = 100 per minute
 - (2) Technique: heel of one hand located over lower half of sternum
 - (3) Compression/ventilation ratio 15:2 (two-rescuer CPR)
 - (4) Depth = at least $\frac{1}{3}$ the total depth of the chest or 2 inches (5 cm) in most children
 - d. Children >8 years old
 - (1) Rate = 100 per minute
 - (2) Technique: heel of one hand with other hand on top located over lower half of sternum
 - (3) Compression/ventilation ratio 15:2 (two-rescuer CPR)
 - (4) Depth = at least $\frac{1}{3}$ the total depth of the chest or more than 2 inches (5 cm) in adolescents
 - e. When two or more healthcare providers are not available, the universal 30:2 compression/ventilation ratio should be used.
 - f. CPR with an advanced airway in place: 8–10 breaths per minute and compressions at 100 per minute

E. Defibrillation and cardioversion

1. Paddle size
 - a. Infants (<10 kg): 4.5 cm
 - b. Children >1 year old or >10 kg: adult paddles 8–13 cm
2. Conducting medium
 - a. Use electrode cream (or paste) or self-adhesive defibrillation pads.
 - b. Do not use alcohol pads; they can produce significant burns.
 - c. Do not allow the conducting medium under one paddle to come into contact with the conducting medium under the other paddle; this can result in “bridging” and delivery of an insufficient amount of current to the heart.

3. Positioning
 - a. Right upper-left lower anterior chest or anterior-posterior orientation: pads should not touch and should be positioned so that there is at least 3 cm between them.
 - b. Infants may need anteriorposterior positioning.
4. Defibrillation
 - a. Initial dosage is 2–4 joules/kg; if unsuccessful, use 4 joules/kg for subsequent attempts, increasing up to 10 joules/kg (maximum adult dosage).
 - b. **If unresponsive with shockable rhythm (ventricular fibrillation or pulseless ventricular tachycardia), initiate CPR before first shock and resume CPR immediately for 2 minutes before checking pulse. Administer epinephrine before proceeding with additional defibrillation attempts.**
5. Cardioversion: initial dosage is 0.5–1 joule/kg.; if unsuccessful, increase the dosage, up to 24 joules/kg.

F. Vascular access

1. Although the tracheal route may be used to administer a limited number of medications, the IV and intraosseous routes are preferable for drug delivery and are mandatory for fluid therapy.
2. The preferred site of vascular access is the *largest* vein that can be rapidly cannulated without disrupting the resuscitation.
 - a. Peripheral venous access (arm, hand, leg, foot) is usually attempted first.
 - b. Central venous access has some advantages and should be used if already in place, but it is more difficult to accomplish in the arrest setting and may be associated with significant complications.
 - c. Administration of drugs via the peripheral venous route should be followed by a saline flush of ≥ 5 mL to promote movement of the drug into the central circulation.
3. **If peripheral venous access cannot be rapidly obtained (3 attempts or 90 seconds) in a child with a life-threatening condition, intraosseous access should be considered. This route is usually easier (more successful and faster) than other types of access in volume-depleted children. In addition, intraosseous access is no longer restricted to young children; it is recommended for the entire spectrum of pediatric care.**
 - a. The anteromedial aspect of the proximal tibia (1–3 cm below the tibial tuberosity on the medial flat surface of the tibia) is the preferred site.
 - b. Drugs, fluids, and blood products may be given by this route. The initial aspirate can also be used for blood typing and chemistries.
 - c. Drug administration should be followed by a saline flush of 5 mL to promote movement of the drug into the central circulation.
 - d. Alternative sites for intraosseous placement include distal femur, medial malleolus, proximal humerus, sternum, and anterior superior iliac spine.
4. The tracheal route is less reliable and therefore less desirable; the kinetics of tracheal drug absorption do not favor this route.
 - a. Medications that are safe and effective when given through the tracheal tube can be remembered by the mnemonic "LEAN":
 - Lidocaine
 - Epinephrine
 - Atropine
 - Naloxone

- b. When this route is used, the dosage administered should be greater than that given by the IV route. However, a specific tracheal dosage has been established only for epinephrine (0.1 mg/kg; 10 times venous dosage), and most authors recommend that the dosage be 2–3 times the IV dosage for other medications.
 - c. Drugs administered by this route should be followed by a 2–5 mL saline flush (or diluted in 2–5 mL of saline) and followed by several positive-pressure ventilations.
 - d. Drug administration should be switched to the IV route as soon as it becomes available.
 - e. Sodium bicarbonate and calcium chloride should not be administered via the tracheal route.
5. In the neonatal period, the umbilical vein is an excellent site of vascular access; it can be used up to 7 days after delivery.

G. Life-threatening cardiac arrhythmias

1. Unlike in adults, in whom rhythm disturbances are primary cardiac diseases, in children rhythm disturbances are frequently secondary problems.
 - a. Most commonly secondary to respiratory (hypoxemia), metabolic/electrolyte, renal disturbances, or drug/toxin effects
 - b. Exceptions: children with congenital heart disease, structural heart disease, or acquired heart disease
2. When evaluating and managing life-threatening dysrhythmias, always consider and treat causes and contributing factors ("6 H's and 5 T's")
 - a. Hypovolemia
 - b. Hypoxia
 - c. Acidosis (hydrogen ion): sodium bicarbonate (1 mEq/kg IV or intraosseous)
 - (1) No longer first-line agent for acidosis
 - (2) Indicated for severe refractory acidosis (after adequate ventilation/oxygenation established and epinephrine ineffective)
 - (3) In infants and children, use 8.4% solution (1 mEq/mL).
 - (4) In neonates, dilute 8.4% solution to make 4.2%.
 - d. Hypokalemia/hyperkalemia
 - (1) Calcium chloride (for life-threatening hyperkalemia with dysrhythmia)
 - (a) 20 mg/kg (0.2 mL/kg) slow IV or intraosseous
 - (b) Preferably should be administered via a central line
 - (2) NaHCO_3 , glucose and insulin, and sodium polystyrene are usually a part of the management of hyperkalemia.
 - e. Hypoglycemia: glucose (0.5–1 g/kg IV or intraosseous)
 - (1) <2 months old: 5–10 mL/kg of D10 solution
 - (2) 2 months to 2 years old: 2–4 mL/kg of D25 solution
 - (3) >2 years old: 1–2 mL/kg of D50
 - f. Hypothermia
 - g. Toxins, poisons, or drugs: naloxone
 - (1) Dosage: 0.1 mg/kg IV push or intraosseous (2 mg IV push or intraosseous if >20 kg) for opioid intoxication
 - (2) Use with caution in neonates, because it can precipitate life-threatening withdrawal in this age group.

- h. Cardiac tamponade
 - i. Tension pneumothorax
 - j. Acute coronary syndrome/pulseless electrical activity (thrombosis)
 - k. Trauma
3. Bradyarrhythmias (most common dysrhythmia seen in pediatric arrest)
 - a. General principle: outside of the immediate neonatal period, only an emergency requiring intervention if causing cardiopulmonary compromise
 - b. Evaluate and support ABCs, including supplemental oxygen.
 - c. If pulse <60 beats per minute and perfusion is poor despite adequate oxygenation and ventilation, start CPR.
 - d. **First drug of choice: epinephrine 0.01 mg/kg (1:10,000) IV or intraosseous or 0.1 mg/kg (1:1,000) if via endotracheal tube; maximum dosage 1 mg IV or intraosseous, or 2.5 mg via endotracheal tube; may repeat every 3–5 minutes**
 - (1) **Epinephrine is the only drug that has been shown to improve return of spontaneous circulation in pediatric arrests. High-dose epinephrine is not recommended.**
 - (2) Efficacy may be reduced by severe acidosis and hypoxia.
 - (3) Continuous infusion of epinephrine (0.1–1 mcg/kg/min IV) may be considered for persistent bradycardia
 - e. Atropine
 - (1) Dosage: 0.02 mg/kg IV or intraosseous (minimum 0.1 mg; maximum 0.5 mg/dose in children and 1 mg/dose in adolescents)
 - (2) Indicated if increased vagal tone or primary AV block is suspected
 - (3) May repeat once with exception of anticholinergic toxidrome/overdose, in which case give every 20–30 minutes until symptoms reversed or secretions dry
 - f. If persistently bradycardic with poor perfusion despite above interventions, consider cardiac pacing.
 4. Tachyarrhythmias (with pulse but poor perfusion)
 - a. Evaluate and support ABCs, including supplemental oxygen and cardiac monitoring.
 - b. Evaluate QRS morphology.
 - (1) Narrow QRS (<90 milliseconds)
 - (a) Sinus tachycardia: evaluate and treat causes.
 - (b) Supraventricular tachycardia
 - i. Infants: heart rate >220 beats per minute
 - ii. Children: heart rate >180 beats per minute
 - iii. Adenosine 0.1 mg/kg IV push (maximum 6 mg); if unsuccessful, 0.2 mg/kg IV push (maximum 12 mg)
 - (2) Wide QRS (>90 milliseconds): ventricular tachycardia
 - (a) Synchronized cardioversion: 0.5–1 joules/kg; if unsuccessful, increase dosage to 2 joules/kg.
 - (b) Amiodarone 5 mg/kg IV over 20–60 minutes or
 - (c) Procainamide 15 mg/kg IV over 30–60 minutes
 5. Pulseless arrest (ventricular tachycardia/ventricular fibrillation, pulseless electrical activity, asystole)
 - a. Start CPR, including rescue breathing, cardiac monitor, IV access.

- b. Evaluate rhythm morphology.
 - (1) Ventricular tachycardia/ventricular fibrillation
 - (a) Defibrillation
 - i. 2–4 joules/kg; if unsuccessful, then 4 joules/kg up to 10 joules/kg (maximum adult dosage) for all subsequent shocks.
 - ii. Always perform CPR for 2–3 minutes immediately after any defibrillation attempts before rechecking rhythm.
 - (b) Epinephrine
 - (c) Consider antiarrhythmic
 - i. Amiodarone (5 mg/kg IV or intraosseous)
 - ii. Lidocaine (1 mg/kg IV or intraosseous; may give second dose of 1.5 mg/kg if unsuccessful)
 - iii. Magnesium (25–50 mg/kg IV or intraosseous) for torsades de pointes

III. UNIQUE ISSUES IN NEONATES AND INFANTS

A. Risk factors associated with neonatal cardiopulmonary arrest

- 1. Maternal factors
 - a. Poor prenatal care
 - b. Age (<16 or >35 years old)
 - c. Preeclampsia/eclampsia
 - d. Hypertension
 - e. Diabetes
 - f. Drug abuse
 - g. Medications (lithium, β -blockers, magnesium)
 - h. Anemia
 - i. Renal disease
 - j. Rh factor incompatibility
 - k. History of prior perinatal morbidity/mortality
 - l. Oligohydramnios
 - m. HIV infection
- 2. Intrapartum factors
 - a. Premature or prolonged labor
 - b. Abnormal presentation
 - c. Precipitous or forceps delivery
 - d. Cephalopelvic disproportion
 - e. Cesarean section
 - f. Cord prolapse or compression
 - g. Analgesia and/or sedatives administered ≤ 2 hours before delivery
 - h. Signs of fetal distress on fetal monitoring (late decelerations)
 - i. Maternal shock

- j. Placenta previa/abruptio
- k. Premature rupture of membranes
- 3. Fetal factors
 - a. Prematurity and postmaturity
 - b. Thick meconium
 - c. Congenital infection
 - d. Malformations identified by ultrasound
 - e. Multiple gestations
 - f. Bradycardia
 - g. Acidosis
 - h. Small fetus for maternal dates
- 4. Precipitous/imminent delivery of newborn
 - a. History
 - (1) Gestational age
 - (2) Prenatal care
 - (3) Single or multiple gestations
 - (4) Color of amniotic fluid
 - b. Equipment
 - (1) Resuscitation bag
 - (2) Neonatal resuscitation masks
 - (3) Dry towels
 - (4) Ambient warmer (if available)
 - (5) Suction equipment (including meconium aspiration device)
 - (6) Uncuffed entotracheal tubes (sizes 2.5, 3.0, and 3.5)
 - (7) Laryngoscope (Miller size 0 and 1)

B. APGAR scoring

Table 27: APGAR Score

	Score		
	0	1	2
Activity (muscle tone)	Limp	Decreased flexion	Good flexion
Pulse (heart rate)	Absent	<100 beats per minute	>100 beats per minute
Grimace (reflex irritability, stimulus respiration)	None	Some motion	Cry
Appearance (color)	Blue, pale	Body pink, extremities blue	Pink
Respirations	Absent	Slow, irregular	Good, crying

1. The Apgar score is assigned to newborns 1–5 minutes after delivery. If the 5-minute score is <7, additional scores are obtained.
2. The Apgar score is helpful in evaluating the newborn's condition and also has a prognostic value. It should not, however, be used as a guide for starting resuscitation—1 minute is far too long to wait. Instead, this decision should be based on the newborn's heart rate

and respiratory status alone; if these are depressed, resuscitation should be started. This includes maintaining temperature, clearing the airway, providing supplemental oxygen, and starting ventilation at 40–60 breaths per minute and CPR (if the heart rate is <60 beats per minute despite adequate ventilation with 100% oxygen for 30 seconds).

C. Meconium staining

1. Occurs in 0.5%–20% of all births
2. Aspiration of thick meconium has a mortality rate of 20%–50%.
3. Routine suctioning of the oropharynx with a bulb syringe before delivering the shoulders has been shown to be of no value. If the fluid contains meconium and the infant has absent or depressed respirations, decreased muscle tone, or a heart rate <100 beats per minute, perform direct laryngoscopy immediately after birth for suctioning of meconium.
4. There is evidence that tracheal suctioning of the vigorous infant with meconium-stained fluid does not improve outcome and may cause complications.

D. Neonatal seizures

1. Types
 - a. Subtle
 - b. Tonic (present as decorticate or decerebrate posturing)
 - c. Focal clonic
 - d. Multifocal clonic
 - e. Myoclonic
2. Etiology
 - a. Hypoxic-ischemic encephalopathy (most common)
 - b. Intracerebral hemorrhage
 - c. Meningitis/encephalitis (toxoplasmosis, syphilis, rubella syndrome, cytomegalovirus, herpes simplex infection ["TORCH" infections])
 - d. Pyridoxine deficiency
 - e. Stroke
 - f. Drug withdrawal
 - g. Developmental anomalies
 - h. Hypoglycemia
 - i. Electrolyte imbalance (\downarrow Ca^{++} , \uparrow Mg^{++} , \downarrow Na^+ , or \uparrow NH_3^+)
 - j. Inborn errors of metabolism (often presenting with \uparrow NH_3^+ , \downarrow glucose, \uparrow lactate, ketonuria, and acid-base disturbances)
3. Management
 - a. Assess ABCs: maintain airway, provide supplemental oxygen, establish an IV line (normal saline), and start appropriate monitoring.
 - b. Correct underlying metabolic problems if present (hypoglycemia, hypocalcemia, electrolyte imbalance).
 - c. Identify and treat associated problems (sepsis, acidosis, etc).
 - d. Begin anticonvulsant therapy.
 - (1) Phenobarbital is the initial anticonvulsant agent of choice in neonates. Dosage is 15–20 mg/kg IV over 10 minutes; if this is ineffective, additional dosages of 5 mg/kg may be given every 5 minutes up to a total maximal dosage of 40 mg/kg.

- (2) If seizures persist or recur, follow with phenytoin.
 - (a) Dosage is 20 mg/kg IV; dilute in normal saline and administer slowly at 0.5 mg/kg/min.
 - (b) Fosphenytoin is another alternative.
- (3) If there is still no response, a benzodiazepine (lorazepam or diazepam) may be given. However, benzodiazepines are not first-line agents in this age group; they produce prolonged and profound respiratory depression (particularly if used in combination with phenobarbital), and they also displace bilirubin from albumin.
 - (a) Lorazepam 0.05–0.1 mg/kg at a rate of 2 mg/min
 - (b) Diazepam 0.2–0.3 mg/kg at a rate of 1 mg/min
- e. Administer pyridoxine 50–100 mg IV or IM for refractory seizures.

E. Congenital diaphragmatic hernia

1. Hernia results from developmental failure of the posterolateral or retrosternal portions of the diaphragm.
2. Left-sided hernias (Bochdalek) are far more common (70%–85%) than right-sided hernias (Morgagni).
3. Associated anomalies include congenital heart disease, GI and genitourinary anomalies, hydronephrosis, and cystic kidneys.
4. Clinical presentation
 - a. History: respiratory distress and vomiting, which is caused by herniation of the abdominal viscera into the chest cavity
 - b. Physical examination: scaphoid abdomen, absence of breath sounds on affected side of chest, auscultation of bowel sounds over affected side of chest
5. Chest radiograph: air-filled loops of bowel in the chest, absence of diaphragmatic margin, displacement of heart and mediastinum, hypoplastic lungs
6. Management
 - a. Immediate intubation (avoid bag-mask ventilation)
 - b. Placement of an orogastric tube, IV hydration, and surgery
7. Mortality rate is 50% in severely affected infants; morbidity is even higher.

F. Tracheoesophageal fistula

1. One-third of affected infants are born prematurely.
2. A proximal esophageal pouch and fistula between the trachea and the distal esophagus (tracheoesophageal fistula) is most common (84%).
3. Associated anomalies and polyhydramnios are common.
4. Infants may present with increased oral secretions, choking, or coughing with attempts at feeding or recurrent aspiration pneumonia.
5. Inability to pass a catheter into the stomach confirms the diagnosis.
6. Management: reverse Trendelenburg or semi-Fowler position, placement of suction catheter into esophageal pouch, IV fluids, and surgical correction

G. Omphalocele and gastroschisis

1. Abdominal wall defects resulting from developmental anomalies of the intestines
 - a. Omphalocele
 - (1) Defect in the umbilical ring with protrusion of the intestines (covered by the peritoneal sac) outside of the abdominal wall

- (2) Associated anomalies (particularly chromosomal ones) occur in one-third to one-half of these infants.
- b. Gastroschisis
 - (1) Defect in the abdominal wall with antenatal evisceration of abdominal contents without a peritoneal sac
 - (2) Occurs in association with intestinal atresia
- 2. Management
 - a. Keep the child warm.
 - b. Place an orogastric tube to decompress the gut.
 - c. Cover the eviscerated intestines with saline-soaked sterile gauze and place them in a plastic bag.
 - d. Administer IV fluids and prophylactic antibiotics.
 - e. Obtain immediate pediatric surgery consultation for operative repair.

H. Necrotizing enterocolitis

- 1. Primarily affects premature infants with very low birth weights
- 2. Characterized by variable degrees of mucosal or transmucosal necrosis of the intestines
- 3. Risk factors
 - a. Hypertonic feeding solutions or medicines
 - b. Patent ductus arteriosus and apneic spells
 - c. Infection
 - d. Ischemia after exchange transfusions
- 4. Onset usually in first 2 weeks of life
- 5. Clinical presentation
 - a. Feeding intolerance
 - b. Abdominal distention with gastric retention (first and most frequent finding)
 - c. Bilious emesis
 - d. Guaiac positive or grossly bloody feces
 - e. Abdominal wall tenderness or erythema
 - f. Manifestations of associated sepsis
 - (1) Metabolic acidosis
 - (2) Apneic episodes
 - (3) Temperature instability
 - (4) Lethargy
- 6. Radiographic findings on plain abdominal radiographs
 - a. Pneumatosis intestinalis (radiographic hallmark)
 - b. Separation of bowel loops (suggests mural edema)
 - c. A fixed, dilated loop of bowel that fails to move on serial radiographs
 - d. Air-fluid levels
 - e. Portal vein gas (sign of severe disease)
 - f. Pneumoperitoneum (indicative of perforation)

7. Management

- a. Discontinue oral feedings.
- b. Place a nasogastric tube to decompress the bowel.
- c. Obtain cultures (blood, feces, urine, CSF).
- d. Administer IV fluids, parenteral feedings, and broad-spectrum antibiotics.
- e. Obtain surgical consult.

I. Cyanosis

1. Central

- a. Bluish discoloration of tongue, mucous membrane, and peripheral skin; unsaturated hemoglobin usually 5 g
- b. Pathologic if persists for >20 minutes after delivery
- c. Etiology
 - (1) Cyanotic heart disease with a right-to-left shunt (the 5 "T's")
 - (a) Transposition of the great vessels
 - (b) Tricuspid atresia
 - (c) Truncus arteriosus
 - (d) Tetralogy of Fallot
 - (e) Total anomalous pulmonary venous return
 - (2) Primary lung disease
 - (3) Hypoventilation due to a CNS lesion (eg, severe intracerebral hemorrhage)
 - (4) Alveolar hypoventilation secondary to shock or sepsis
 - (5) Methemoglobinemia
- d. The "hyperoxia test" (response of the PaO_2 to the administration of 100% oxygen) can provide a diagnostic clue as to the underlying cause of the central cyanosis.
 - (1) Failure of the PaO_2 to rise above 100 mmHg suggests either cyanotic heart disease with a fixed right-to-left shunt or methemoglobinemia.
 - (2) Improvement in the PaO_2 suggests one of the other causes (eg, lung disease, sepsis, CNS disorder).

2. Peripheral

- a. Bluish discoloration of extremities only; oxygen saturation >94%
- b. Common in newborns in the first few days of life
- c. Generally due to vasomotor instability secondary to a cold environment

J. Congestive heart failure

1. Clinical presentation

- a. Feeding difficulty (slow feeder, diaphoresis, and dyspnea on feeding)
- b. Tachypnea and tachycardia
- c. Rales and rhonchi
- d. Hepatomegaly and cardiomegaly
- e. Failure to thrive
- f. Peripheral edema rare

2. Etiology
 - a. Usually due to congenital heart disease, most commonly left-to-right shunts that present at 6–8 weeks
 - b. Other causes include severe anemia, dysrhythmias (supraventricular tachycardia, ventricular tachycardia), sepsis, and arteriovenous malformation (hepatic, cerebral).
3. Management
 - a. Supplemental oxygen
 - b. Semireclining position
 - c. Restriction of fluid intake
 - d. Digoxin
 - (1) The oral digitalizing dosage is 10–20 mcg/kg for preterm infants and 30 mcg/kg for term infants; the IV dosage is three-fourths of the oral dosage.
 - (2) Administer one-half of this dose initially, one-fourth in 8 hours, and the last one-fourth 8 hours later.
 - (3) Contraindications: tetralogy of Fallot, idiopathic hypertrophic subaortic stenosis and myocarditis (a relative contraindication), and a heart rate <100 beats per minute
 - e. Furosemide 1–3 mg/kg IV
 - f. Inotropic support with dopamine and dobutamine may be needed in patients with cardiogenic shock. Afterload reduction may also be indicated.

K. Jaundice in the newborn

1. Physiologic jaundice
 - a. Hyperbilirubinemia that occurs in nearly all newborns as a result of changes in bilirubin metabolism resulting from increased production, decreased clearance, and increased enterohepatic circulation
 - b. Always unconjugated (indirect) hyperbilirubinemia
 - c. Resolves within 1–2 weeks
2. Pathologic jaundice
 - a. Hyperbilirubinemia greater than the 95th percentile for hours-of-age
 - b. Risk factors/causes for severe hyperbilirubinemia
 - (1) Blood type (A, B, O) or Rh incompatibility (Coombs' test positive)
 - (2) Glucose-6-phosphate dehydrogenase deficiency (or other RBC membrane abnormalities)
 - (3) Preterm or late preterm delivery
 - (4) Birth trauma (large cephalohematomas)
 - (5) Infants who are breast-fed only
 - (6) Sepsis
 - c. Clinical presentation
 - (1) Lethargy or irritability
 - (2) Poor feeding
 - (3) Dehydration
 - (4) Hypotonia
 - (5) Seizures

- d. Management
 - (1) IV hydration
 - (2) Treat any underlying pathology.
 - (3) Phototherapy and exchange transfusion for severe hyperbilirubinemia: based on total serum bilirubin gestational age and hours of life (published guidelines exist) but concerning if >20 mg/dL
- e. Complication of acute bilirubin encephalopathy (kernicterus): neurologically devastating sequelae of bilirubin crossing the blood-brain barrier, resulting in choreoathetoid cerebral palsy, sensorineural hearing loss, and gaze palsies

L. Apparent life-threatening event (ALTE)

1. Diagnostic criteria: acute unexpected change in an infant's breathing behavior accompanied by one or more of the following:
 - a. Change in muscle tone (hyper- or hypotonia)
 - b. Change in color (cyanosis, pallor, dusky, dark red)
 - c. Choking or gagging
 - d. Caregiver has perception that episode was life-threatening.
2. Most children presenting with ALTEs have normal physical examinations in the emergency department.
3. Differential diagnosis
 - a. Gastroesophageal reflux disease (most common in feeding-related episodes)
 - b. Seizures
 - c. Upper airway obstruction
 - d. Ingestions
 - e. Child abuse (usually with head injuries)
 - f. Dysrhythmias
 - g. Sepsis (occult systemic bacterial illness presenting as ALTE in the afebrile, well-appearing infant is rare)
4. Diagnostic evaluation (as indicated by history and physical examination)
 - a. CBC
 - b. Blood chemistries (electrolytes, glucose)
 - c. Cultures (blood, urine, CSF)
 - d. Chest radiograph
 - e. ECG
 - f. Head CT, skull radiographs
 - g. Toxicologic screen
 - h. Lumbar puncture
5. Additional inpatient evaluation
 - a. Ammonia level
 - b. Feces for *Clostridium* culture and botulinum toxin
 - c. Electroencephalogram
 - d. Apnea monitoring
 - e. Barium swallow

- f. 24-hour cardiac monitoring
- g. Polysomnography
- 6. Management
 - a. ABCs
 - b. Treat any underlying pathology
- 7. Disposition: all infants with ALTEs should be admitted for monitoring/observation and any additional diagnostic evaluation.
- 8. Prognosis
 - a. Overall risk of subsequent death in infants diagnosed with ALTE is <1%.
 - b. Certain subgroups have higher rates of subsequent sudden infant death syndrome
 - (1) Recurrent ALTEs requiring CPR
 - (2) Premature infants
- 9. Periodic breathing of infancy
 - a. *Normal* breathing pattern of newborns (especially premature neonates) that is often mistaken for ALTEs
 - b. Characterized by periods of rapid breathing followed by apnea of 3–20 seconds in duration
 - c. Never associated with cyanosis or bradycardia

M. Sudden infant death syndrome

1. Although the incidence in the United States has been reduced by >50% (down to 3,700 deaths per year) as a result of the "back to sleep" initiative, which encouraged parents to place sleeping infants in the supine position, sudden infant death syndrome remains the leading cause of death in infants 1 month to 1 year old.
2. Peak incidence is during the winter months during the hours of sleep (midnight to 9 am) in infants 2–4 months old; boys are more commonly affected than girls.
3. Although the exact cause(s) remains unclear, current pathologic data indicate that hypoxia (and possibly autonomic dysfunction) plays a role in the pathophysiology.
4. Risk factors
 - a. Maternal
 - (1) Age <20 years at time of the first pregnancy (one of two highest risk factors)
 - (2) Smoker (one of two highest risk factors)
 - (3) Short interpregnancy intervals
 - b. Prenatal
 - (1) Prematurity
 - (2) Low birth weight for gestational age
 - (3) Intrauterine growth retardation
 - c. Postnatal
 - (1) Sleeping in the prone position
 - (2) Soft bedding
 - (3) Parent sleeping with infant

IV. CONGENITAL HEART DISEASE

A. Cyanotic heart lesions

1. Diagnostic clue to the presence of these lesions: pulse oximetry reveals desaturation and does not improve significantly in response to administration of 100% oxygen (hyperoxia test: 100% oxygen administered for 5 minutes and the $\text{PaO}_2 < 100$).
2. Tetralogy of Fallot
 - a. Most common type of cyanotic congenital heart disease in children >1 year old
 - b. The four anatomic components of tetralogy of Fallot
 - (1) Ventricular septal defect
 - (2) Pulmonic stenosis
 - (3) Dextroposition and overriding of the aorta
 - (4) Right ventricular hypertrophy
 - c. The two major hemodynamic problems associated with tetralogy of Fallot
 - (1) The ventricular septal defect permits right-to-left shunting.
 - (2) The severity of the pulmonic stenosis determines the degree of cyanosis from right-to-left shunting through the ventricular septal defect.
 - d. Clinical presentation
 - (1) Cyanosis
 - (2) Clubbing of the digits
 - (3) Normal pulses
 - (4) Harsh, diamond-shaped systolic murmur of pulmonic stenosis heard best in the second intercostal space along the left sternal border
 - (5) Loud, single second heart sound
 - (6) Patients develop "hypercyanotic" ("Tet") spells with moderate to severe pulmonic stenosis. They are most common in the first 3 years of life and are brought on by exertion (feeding, straining, crying). These episodes are treated by placing the child in the prone knee-chest position and administering supplemental oxygen and morphine 0.1 mg/kg IV, IM, or SC. The morphine may be repeated if the child does not respond to the initial dose. If there is still no response, propranolol (0.05–0.1 mg/kg IV) or phenylephrine (10 mcg/kg IV followed by 0.5–2 mcg/kg/min) may be given under the direction of appropriate pediatric consultation.
 - e. Diagnostic evaluation
 - (1) Chest radiograph findings
 - (a) Boot-shaped heart
 - (b) Diminished pulmonary vascular markings (due to decreased pulmonary blood flow)
 - (2) ECG findings
 - (a) Right axis deviation
 - (b) Right ventricular hypertrophy
3. Other causes of cyanotic heart disease
 - a. Transposition of the great vessels (most common cause in newborns)
 - b. Total anomalous pulmonary venous return
 - c. Truncus arteriosus

- d. Tricuspid atresia
- e. Pulmonary atresia
- f. Ebstein anomaly of the tricuspid valve

B. Acyanotic heart lesions

1. Aortic stenosis (most common)

- a. A bicuspid aortic valve is the most frequent cause of significant valvular aortic stenosis in infancy and childhood; it is one of the most common congenital heart lesions identified in adults.
- b. Although generally seen in older children, severe lesions may present in infancy as left heart failure. Critical stenosis results in heart failure and cardiogenic shock when the ductus arteriosus closes.
- c. Clinical presentation
 - (1) Exercise intolerance
 - (2) CHF
 - (3) Chest pain
 - (4) Syncope
 - (5) Diamond-shaped systolic ejection murmur radiating to the neck
 - (6) Systolic ejection click
 - (7) An S_3 is common; an S_4 indicates severe stenosis.
 - (8) Palpable thrill over the base of the heart
- d. ECG findings: signs of left ventricular hypertrophy or strain may be present
- e. Complications
 - (1) Infectious endocarditis
 - (2) Sudden death due to ventricular dysrhythmias

2. Ventricular septal defect

- a. Most common congenital heart anomaly
- b. Clinical presentation is determined by the size of the lesion.
 - (1) Small defects: may be asymptomatic
 - (2) Large defects: signs of CHF (tachypnea, grunting respirations, fatigue with feeding) typically develop after the first few weeks or months of life as the pulmonary vascular resistance falls and flow across the defect increases.
 - (3) Physical findings
 - (a) Holosystolic murmur at the lower left sternal border
 - (b) Normal peripheral pulses

3. Other causes of acyanotic heart disease

- a. Atrial septal defect
- b. Patent ductus arteriosus (common in premature infants): characterized by bounding pulses, a continuous "machinery" murmur, and a suprasternal notch thrill
- c. Coarctation of the aorta: cardinal findings are hypertension in the upper extremities and decreased or absent pulses in the lower extremities.

C. Ductal dependent lesions

- 1. Cardiac lesions that manifest as outflow tract obstruction (severe coarctation of the aorta, critical aortic stenosis, hypoplastic left heart syndrome, and tricuspid atresia)

2. Infants with these lesions usually present in profound shock with a classic skin color described as the "gray baby" within hours to days of delivery.
3. Affected infants become symptomatic as the ductus arteriosus closes, because the systemic circulation (in left-sided lesions) or pulmonic circulation (in right-sided lesions) depends on shunting.
4. Management
 - a. ABCs, IV line, monitor
 - b. Prostaglandin E_1 , a potent dilator of the ductus arteriosus, can be life-saving in these infants. The infusion (0.05–0.1 mcg/kg/min) should be started, and the child monitored for apnea and hypotension (occurs with prostaglandin infusion).
 - c. Obtain urgent cardiology consult.

D. Congestive heart failure

1. Primary cause in infants and young children is congenital heart disease.
2. Etiology based on age of presentation:
 - a. Newborn (noncardiac causes)
 - (1) Anemia
 - (2) Hypoxia
 - (3) Acidosis
 - (4) Hypoglycemia
 - (5) Hypocalcemia
 - (6) Sepsis
 - b. Day 1 → patent ductus arteriosus
 - c. First week → hypoplastic left heart syndrome
 - d. 2 weeks → coarctation of the aorta
 - e. 1 month → ventricular septal defect
 - f. 3 months → supraventricular tachycardia (the most common symptomatic dysrhythmia in infants and children)
 - g. 1–2 years
 - (1) Myocarditis
 - (2) Cardiomyopathy
 - (3) Severe anemia
 - (4) Kawasaki disease
 - h. 10 years → rheumatic fever
3. Clinical presentation
 - a. Right-sided CHF → hepatomegaly
 - b. Left-sided CHF → tachypnea, dyspnea, diaphoresis, poor feeding
 - c. Both → cardiomegaly, tachycardia, cyanosis, failure to thrive
4. Diagnostic evaluation: chest radiograph typically shows cardiomegaly and increased pulmonary marking.
5. Treatment: see management of newborn (see page 639)

V. AIRWAY EMERGENCIES

A. Upper airway (see also upper airway emergencies, page 190)

1. Clinical presentation
 - a. Stridor
 - (1) Inspiratory (obstruction at or above larynx)
 - (2) Biphasic (obstruction below the larynx)
 - (3) Expiratory (bronchial or lower tracheal obstruction)
 - b. Expiratory wheezing (obstruction below the carina at the level of the bronchi)
 - c. Tachypnea
 - d. Nasal flaring
 - e. Intercostal and substernal retractions
 - f. Cyanosis
 - g. Hoarseness
 - h. Coughing
 - i. Grunting and expiratory wheezing are signs of *lower* airway distress.
2. Foreign body aspiration
 - a. Most common cause of accidental home death in children <6 years old
 - b. If stridor is present, the foreign body is in the larynx or trachea; if wheezing is present, the foreign body is in a mainstem bronchus or further down the bronchial tree.
 - c. Most foreign bodies are located in a mainstem bronchus (frequently the right) and are associated with a triphasic course of symptoms.
 - (1) Acute onset of symptoms (choking, coughing, gagging)
 - (2) Latent asymptomatic period as the foreign body passes into the smaller airways
 - (3) Delayed onset of wheezing or stridor (depending on location), followed by pneumonia
 - d. Recurrent pneumonia at the same location in a child is highly suspicious of an occult foreign body aspiration.
 - e. Diagnostic evaluation
 - (1) A radiopaque foreign body (eg, a coin) located in the trachea is seen on edge in the PA view of the chest (and "en face" in the lateral view), while one located in the esophagus is seen "en face" in the PA view (and on edge in the lateral view).
 - (2) With a foreign body that is partially obstructing a mainstem bronchus, the obstructed lung is hyperinflated (ball-valve effect) with a mediastinal shift away from the foreign body on the expiratory view.
 - (3) Bilateral decubitus radiographs can be done in young, uncooperative children; look for paradoxical hyperinflation.
 - (4) With complete obstruction, the involved lung becomes atelectatic.
 - (5) MRI can be useful in confirming the presence of a peanut or other vegetable matter.
 - f. Management: removal via bronchoscopy
3. Epiglottitis/supraglottic (usual age group 2–6 years old)
 - a. Relatively uncommon since the introduction of the *Haemophilus influenzae* B vaccine; accordingly, epiglottitis is most commonly seen in unvaccinated children or adults today.

- b. Clinical presentation: A child is sitting upright with chin thrust forward, mouth open, and neck slightly extended. He appears toxic and apprehensive and, on closer inspection, is drooling, with difficulty swallowing and stridor. The mother relates that the child was well until just a few hours ago, when he started to complain of a sore throat (or just not feeling well) and developed a fever. A cough is usually absent.
 - c. Management
 - (1) **It is best to disturb these children as little as possible to reduce anxiety, because they are at high risk of complete airway obstruction. Allow the child to assume a position of comfort (usually sitting upright) and remain in the company of the parents; do not force the child to lie supine.**
 - (2) Establish the diagnosis
 - (a) If there is only slight respiratory distress, the diagnosis can be confirmed with a portable lateral soft-tissue radiograph of the neck. Findings include the "thumb print sign" and mild hypopharyngeal distention. If this study cannot be done portably, someone skilled in airway management must accompany the child to the radiology department with the appropriate invasive airway equipment in hand.
 - (b) Evaluation of the epiglottis by using a tongue blade (or a fiberoptic bronchoscope) can be safely done by experienced physicians, but it remains controversial and should be avoided in children with any respiratory distress.
 - (c) Definitive diagnosis is ideally made in the operating room under controlled conditions by direct visualization of the "cherry-red" epiglottis. This is particularly true when severe respiratory distress is present.
 - (3) Provide supplemental humidified oxygen, set up airway stabilization equipment at bedside (the patient can usually be adequately ventilated with bag and mask if total airway obstruction occurs), and obtain immediate ENT and anesthesia consultation.
 - (4) Ideally, the child should be intubated under general anesthesia in the presence of an ENT surgeon. If unable to intubate, the surgeon will need to perform a tracheostomy.
 - (5) Once the airway has been stabilized, draw blood for laboratory studies (including cultures); blood cultures are positive in 80% of patients.
 - (6) Administer parenteral antibiotics. Acceptable regimens include a second- or third-generation cephalosporin (cefuroxime, cefotaxime, or ceftriaxone) or ampicillin-sulbactam.
 - (7) Admit to ICU.
 - d. Although *Haemophilus influenzae* type B remains the most common etiologic agent, the number of cases caused by this organism has markedly decreased since the advent of the *H influenzae* B vaccine. Group A β -hemolytic streptococci (followed by *Streptococcus pneumoniae* and *Staphylococcus aureus*) are responsible for an increasing number of cases.
4. Croup (usual age group 6 months to 6 years old, peak incidence at 2 years old)
 - a. Clinical presentation: The child has a barking, "seal-like" cough that is usually worse at night and may cause severe respiratory distress. The illness tends to come on gradually (usually in the late fall or early winter) as an upper respiratory infection. The cough, hoarseness, and stridor typically appear 1–2 days later. Croup symptoms peak on the third or fourth day of the illness (which is the first and second day of the croup symptoms) and generally resolve over a week. Fever is absent or low-grade, and the child is nontoxic in appearance.

- b. Diagnostic evaluation
 - (1) Radiographs are not usually needed (diagnosis can often be made on clinical grounds alone) but are useful in excluding epiglottitis.
 - (2) The PA neck radiograph reveals symmetric subglottic narrowing of the tracheal air column ("steep sign"), and the lateral neck radiograph shows a distended hypopharynx and subglottic narrowing. The epiglottis and retropharyngeal space are normal.
- c. Management: supportive
 - (1) Antibiotics are not indicated because the infecting organism is viral (usually parainfluenza virus).
 - (2) Cool mist
 - (3) Oxygen as needed
 - (4) Hydration (orally or IV)
 - (5) Racemic epinephrine aerosol (0.5 mL of a 2.25% solution diluted in 2.5 mL of saline)
 - (a) Reserved for children with resting stridor and respiratory distress
 - (b) Children who receive it should be observed in the emergency department for 3–4 hours after administration in case of return to their pretreatment stridorous state once the effect wears off.
 - (c) If racemic epinephrine is unavailable, aerosolized L-epinephrine 1:1,000 (5 mL) is equally effective and may be used.
 - (6) Steroids (dexamethasone 0.3–0.6 mg/kg IM, IV, or orally as a one-time dose)
 - (a) Should be administered to all children with croup (mild, moderate, or severe) and especially to those who receive racemic epinephrine
 - (b) Hastens symptomatic improvement
 - (7) Heliox (a mixture of helium and oxygen)
 - (a) Decreases the work of breathing by improving laminar gas flow through the obstructed airway
 - (b) Usually reserved for children with severe croup or refractory respiratory distress despite steroids and racemic epinephrine
 - (8) Although most children can be discharged, admission should be considered for those who:
 - (a) Have persistent stridor at rest
 - (b) Are unable to tolerate oral fluids
 - (c) Have an incomplete response to racemic epinephrine or require multiple doses
 - (d) Present with severe croup (particularly those <1 year old)
- 5. Bacterial tracheitis (usual age <3 years old)
 - a. Clinical presentation: The child has a high fever, stridor, and appears toxic. His mother reports that he (or she) has had symptoms of viral croup (nasal congestion, barking cough, stridor) for the past few days and then suddenly took a turn for the worse over the past few hours.
 - b. Etiology: *S aureus* (most common), *S pneumoniae*, and anaerobes
 - c. Management
 - (1) Provide supplemental humidified oxygen.

- (2) Obtain immediate consultation with ENT and anesthesiology for endoscopy. This procedure is performed in the controlled setting of the operating room and confirms the diagnosis. Findings include pseudomembranes and purulent secretions.
 - (3) Once the airway has been secured, appropriate laboratory studies (including a Gram stain and culture of tracheal secretions) should be obtained; IV hydration and antibiotics effective against *S aureus* should be administered.
 - (4) Admit to ICU.
6. Retropharyngeal abscess (usual age group 6 months to 6 years old, peak incidence 3–5 years old)
- a. Clinical presentation: toxic child
 - (1) Incidence declines after 6 years old because the lymph nodes atrophy.
 - (2) In older children, usually secondary to trauma and iatrogenic causes
 - (3) Toxic-appearing child with fever, sore throat, neck stiffness, drooling, and refusing to eat
 - b. Diagnostic evaluation
 - (1) Soft-tissue lateral radiograph of the neck
 - (a) Initial study of choice
 - (b) Must be taken during inspiration with the neck in slight extension to be accurate
 - (c) Reveals a widened retropharyngeal space (the normal width of this space is less than half the width of the adjacent vertebral body)
 - (2) Chest radiograph to exclude the complication of mediastinitis
 - (3) CT or MRI of the neck and mediastinum
 - (a) Confirms the diagnosis
 - (b) Distinguishes between an abscess and cellulitis
 - (c) Establishes the presence and extent of complications
 - c. Etiology: *S aureus*, group A β -hemolytic streptococci, and anaerobes
 - d. Management
 - (1) Broad-spectrum parental antibiotics
 - (a) A penicillinase-resistant penicillin and a third-generation cephalosporin and metronidazole *or*
 - (b) Clindamycin and an aminoglycoside
 - (2) Emergent ENT consultation
 - (3) ICU admission

B. Lower airway

1. Bronchiolitis
 - a. Occurs with greatest frequency in the spring and winter months in infants 2–6 months old
 - b. Etiology: respiratory syncytial virus
 - c. Infection \rightarrow submucosal edema, peribronchiolar cellular infiltrate, and mucous plugging \rightarrow narrowing of the bronchi and bronchioles \rightarrow increased airway resistance \rightarrow wheezing and fine rales
 - d. Clinical presentation

- (1) Upper respiratory symptoms (runny nose, sneezing) before going on to develop lower respiratory symptoms (tachypnea, wheezing)
- (2) Examination reveals a tachypneic infant with nasal flaring, intercostal retractions, wheezing, rales, and a prolonged expiratory phase.
- (3) Cyanosis and signs of dehydration may also be present.
- e. Diagnostic evaluation
 - (1) Chest radiograph reveals hyperinflation of the lungs and patchy atelectasis.
 - (2) Immunofluorescence analysis or ELISA of nasal wash specimens can confirm the presence of respiratory syncytial virus infection and impact subsequent care.
- f. Differential diagnosis includes asthma ("reactive airway disease"), cystic fibrosis, recurrent aspiration, congenital heart disease, and foreign body aspiration. A careful history and chest radiographs are helpful in eliminating some of these diagnoses. However, differentiating between asthma and bronchiolitis can be difficult because of the similarity in presenting symptoms.
- g. Prevention: Prophylactic regimens using immune globulin (palivizumab) are available for high-risk infants (premature infants and those with lung or heart disease) to lessen the risk of subsequent severe respiratory syncytial virus infection.
- h. Risk factors for severe disease
 - (1) Premature infants
 - (2) Congenital heart disease
 - (3) Respiratory syncytial virus subtype A
 - (4) History of cyanosis/apnea
 - (5) Age <3 months or first 3 days of illness
 - (6) Atelectasis on chest radiograph
 - (7) Increased respiratory rate with use of accessory muscles
 - (8) Toxic appearance
- i. Management is primarily supportive.
 - (1) Check oxygenation with a pulse oximeter or arterial blood gases; hypoxia is common and should be treated with humidified oxygen at an FiO_2 of 28%–40%; oxygen saturation <95% (in room air) is the single best predictor of severe bronchiolitis.
 - (2) Children who are dehydrated because of decreased fluid intake should be given IV hydration.
 - (3) Nebulized epinephrine (either racemic or the L-isomer) or nebulized albuterol are controversial in the acute treatment of bronchiolitis. For children with respiratory distress, a trial of nebulized albuterol and/or epinephrine is reasonable.
 - (4) Corticosteroids are not routinely indicated.
 - (5) Antibiotics are indicated only if concomitant bacterial infection is suspected.
 - (6) Positive-pressure ventilation (continuous positive-airway pressure [CPAP] or bilevel positive-airway pressure [BiPAP]) is useful in severe disease)
 - (7) Admission criteria
 - (a) Inability to tolerate fluids
 - (b) Hypoxia or history of apnea or cyanosis
 - (c) Severe respiratory distress
 - (d) Apneic episodes

- (e) Prematurity
 - (f) Lethargy
 - (g) Presence of underlying medical conditions (eg, congenital heart or lung disease, immunodeficiency, or immunosuppression)
 - (h) Unreliable caregiver
 - j. Complications: apnea is a serious complication of respiratory syncytial virus bronchiolitis (18% of infants). Very young (<2 months old) and premature infants are at highest risk; these patients are frequently hospitalized.
2. Asthma (most common chronic disease in childhood)
- a. Epidemiology
 - (1) The prevalence of asthma in the United States is on the rise.
 - (2) Despite improvement in therapy, the incidence of asthma-related deaths is also increasing.
 - b. Definition and etiology
 - (1) Asthma is a chronic inflammatory disorder of the airways. It is characterized by increased responsiveness of the trachea and bronchi to a variety of stimuli ("triggers") and is manifested by partially or completely reversible airway obstruction, increased mucus production, and airway edema.
 - (2) Serum IgE levels and the prevalence of asthma are closely related, lending support to the premise that asthma nearly always has an allergic basis. Further support comes from the fact that atopic individuals (those with a genetic predisposition for producing an IgE-mediated response to common environmental allergens) are at greatest risk of developing asthma.
 - (3) "Triggers" that can precipitate an asthma attack
 - (a) Viral respiratory infections (most frequent trigger in children <2 years old with respiratory syncytial virus being particularly common in preschool-age children and *Mycoplasma* being more common in school-age children)
 - (b) Allergens (eg, dust mite, cockroach, pollens, animal dander, fungi such as *Alternaria*)
 - (c) Exercise
 - (d) Inhaled irritants (cigarette smoke, air pollution, smoke from burning wood, fumes)
 - (e) Medications (eg, aspirin, NSAIDs, β -blockers)
 - (f) Food additives (eg, sulfites, MSG, some dyes)
 - (g) Gastroesophageal reflux
 - (h) Cold exposure and changes in humidity
 - (i) Endocrine factors (eg, menses, pregnancy, thyroid disease)
 - (j) Psychological factors
 - c. Pathophysiology
 - (1) These "triggers" enhance the release of preformed mediators of inflammation (eg, histamine, chemotactic factors) from mast cells \rightarrow bronchial smooth muscle contraction and \uparrow vascular permeability \rightarrow airway edema and increased mucus production followed (in a few hours) by migration of inflammatory cells into the airway and secretion of additional chemotactic and vasoconstrictive products \rightarrow increased bronchial hyperresponsiveness

- (2) Bronchoconstriction occurs initially, followed by the development of airway inflammation (accumulation of inflammatory cells, edema, hypersecretion, epithelial shedding) → airway obstruction and increased airway resistance → air trapping, atelectasis, ventilation-perfusion mismatching, hypoxia, hypercarbia
- d. Clinical presentation
 - (1) Physical examination often reveals signs of respiratory distress (intercostal retractions, nasal flaring, prolonged expiratory phase, cyanosis) as well as tachycardia, tachypnea, and wheezing (which may be absent in the "tight" asthmatic who is not moving much air, ie, "silent chest"). In some patients, however, only a chronic cough or decreased exercise tolerance is present.
 - (2) It is important to evaluate vital signs and lung sounds before and after each therapeutic intervention to see if they are improving or worsening and to determine if any signs of toxicity develop.
- e. Diagnostic evaluation
 - (1) Oxygen saturation should be assessed with pulse oximetry; a low saturation (90%), especially at initial triage, is a poor prognostic indicator, suggesting a need for prolonged observation or admission.
 - (2) Pulmonary function testing: peak expiratory flow rate (PEFR) or forced expiratory volume (FEV₁) should be measured before and after each treatment with an adrenergic agent.
 - (a) These tests assess the degree of functional airway obstruction and provide a measure of the patient's response to therapy.
 - (b) Most children ≥5 years old can successfully perform them (effort dependent).
 - (c) PEFR <50% of predicted indicates a severe exacerbation; PEFR 50%–80% of predicted indicates a moderate exacerbation.
 - (3) Chest radiograph: Although not indicated in all patients, it should be obtained in patients presenting with their first episode of wheezing (to exclude other pathology) and in those who may have complications (atelectasis, pneumothorax, pneumonia, pneumomediastinum). It generally reveals hyperinflation and flattened diaphragms.
- f. Asthma severity: a combination of signs, symptoms, functional assessment parameters (eg, SaO₂, PEFR), and historical patient data can be used to assess the severity of an exacerbation and risk of death from asthma. This information, when combined with the response to treatment measures, is helpful in determining the need for hospital admission. Unlike in children with asthma, the use of asthma indices (eg, the Fischl scoring system) has not proved to be a reliable indicator of asthma severity in adults.
 - (1) Exacerbations are classified as mild, moderate, or severe based on a combination of several parameters.
 - (a) Patients with mild exacerbations
 - i. Speak in complete sentences
 - ii. Have only end-expiratory wheezing
 - iii. Are generally not using accessory muscles
 - iv. Do not have pulsus paradoxus
 - v. Have a PEFR >80% of predicted/personal best and an SaO₂ >95% on room air (at sea level)
 - (b) Patients with moderate exacerbations
 - i. Speak in phrases
 - ii. Have wheezing throughout exhalation

- iii. Commonly use accessory muscles
 - iv. May have a pulsus paradoxus of 10–25 mmHg
 - v. Have a PEFR 50%–80% of predicted/personal best and an SaO_2 of 91%–95% on room air (at sea level)
- (c) Patients with severe exacerbation
- i. Speak one or two words at a time
 - ii. Commonly have loud wheezing throughout inhalation and exhalation
 - iii. Are usually using accessory muscles
 - iv. Frequently have a pulsus paradoxus of 20–40 mmHg
 - v. Have a PEFR <50% of predicted/personal best and an SaO_2 <91% on room air (at sea level)
- (d) A more complete list of the parameters that can be used to classify the severity of an acute exacerbation can be found in NIH guidelines for the Diagnosis and Management of Asthma (Expert Panel Report) (<http://www.nhlbi.nih.gov/guidelines/asthma/asthgdln.pdf>).
- (2) Risk factors for death from asthma
- (a) Past medical history of severe exacerbations (eg, prior intubation or ICU admission for asthma) or those that are abrupt in onset
 - (b) ≥ 2 hospitalizations or ≥ 3 emergency department visits in the past year
 - (c) Hospitalization or emergency department treatment in the last 30 days
 - (d) Current use or recent cessation of oral steroids
 - (e) Use of >2 canisters of a short-acting inhaled β_2 -agonist per month
 - (f) Presence of significant comorbid disease (eg, COPD, coronary artery disease) or psychiatric illness
 - (g) Urban residency and/or lower socioeconomic status
 - (h) Sensitivity to *Alternaria* (mold)
 - (i) Use of illicit drugs
- g. Management
- (1) Administer oxygen to all asthmatic patients; most are hypoxic, and treatment with β -adrenergic agents may initially worsen hypoxia by increasing the ventilation-perfusion mismatch. Maintain oxygen saturation >95% in children and infants.
 - (2) The initial treatment of choice is aerosolized therapy with a β -adrenergic agonist; it is associated with fewer systemic adverse effects and better dilation than parenteral therapy, and it obviates the need for painful injections.
 - (a) Albuterol 0.5% solution (5 mg/mL) is the preferred agent because of its β_2 -agonist specificity.
 - i. Administer 0.15 mg/kg (maximum dose is 5 mg; minimum dose is 2.5 mg) in 2–3 mL normal saline every 20 minutes for 3 doses, then 0.15–0.3 mg/kg up to 10 mg every 1–4 hours as needed.
 - ii. Continuous nebulization using 0.5 mg/kg/hr (maximum 15 mg/hr) should be considered for children who do not respond to this regimen or who present with severe respiratory distress.
 - iii. Albuterol administered by a metered-dose inhaler (90 mcg/puff) with a spacer is as effective as nebulizer therapy and can be used in children who

are able to coordinate the inhalation maneuver (usually children >6 years old). The dosage is 4–8 puffs every 20 minutes up to 4 hours, then every 1–4 hours as needed.

- (b) Alternative agents include terbutaline (parenteral formulation), bitolterol, and pirbuterol. These agents do not appear to provide any clear advantage over albuterol, although pediatric experience with bitolterol and pirbuterol is limited. Because of its lack of β_2 -agonist specificity, metaproterenol is no longer recommended as a first-line agent. Albuterol is also available as an isomer (levalbuterol); compared with albuterol, less frequent dosing may be possible, and it may produce less direct effect on β_1 -adrenergic receptors and/or fewer cardiac adverse effects, but these claims have not been consistently demonstrated in clinical trials.
- (3) Parenteral adrenergic therapy is a useful adjuvant to ongoing aerosolized therapy for the very sick (those with a decreased level of consciousness) and the very young who may not be able to provide the necessary inspiratory effort or cooperate. Alternatives include:
 - (a) Epinephrine (1:1,000 solution)
 - i. 0.01 mg/kg (up to 0.5 mg) SC every 20 minutes for 3 doses
 - ii. IM route has more consistent and rapid absorption than SC route.
 - (b) Terbutaline (1 mg/mL)
 - i. Preferred agent because it is β_2 -selective
 - ii. 0.005–0.01 mg/kg (up to 0.4 mg) SC every 20 minutes for 3 doses, then every 2–6 hours as needed
- (4) Corticosteroids are useful in treating the inflammatory aspect of asthma. When administered early, they prevent progression of the illness and decrease the rate of relapse.
 - (a) Administer these agents as soon as it is clear that they are indicated:
 - i. All patients with moderate to severe exacerbations (administer them along with initial β_2 -agonist therapy)
 - ii. Those patients with mild exacerbations who:
 - Do not clear completely with initial β_2 -agonist therapy
 - Have recently discontinued an oral steroid preparation
 - (b) Dosage
 - i. Methylprednisolone 1–2 mg/kg IV or orally *or*
 - ii. Prednisolone 1–2 mg/kg orally *or*
 - iii. Prednisone 1–2 mg/kg orally *or*
 - iv. Dexamethasone 0.6 mg/kg IV, IM, or orally
 - (c) Liquid prednisone/prednisolone/dexamethasone is absorbed rapidly; therefore, unless the patient is vomiting or in severe distress, there is no advantage to IV administration of corticosteroids.
 - (d) Patients who receive prednisone or prednisolone in the emergency department and are subsequently discharged should be continued on oral steroids at a dosage of 1–2 mg/kg/day \times 5 days. Patients who receive dexamethasone should receive one additional dose in 1–2 days (longer half-life).
- (5) Ipratropium bromide is an inhaled anticholinergic agent that, when used in combination with a β -adrenergic agonist, exhibits an additive effect by reducing

bronchoconstriction and decreasing mucus production. In patients with severe exacerbations, ipratropium should be added to the first three β_2 -agonist treatments; dose is 250–500 mcg via nebulizer or 4–8 puffs via a metered-dose inhaler.

- (a) Ipratropium bromide 0.25 mg/dose in 2 mL normal saline every 20 minutes for three doses, then every 2–4 hours (agent of choice)
- (b) Atropine is associated with a high incidence of adverse effects and is rarely used.
- (6) Magnesium sulfate acts as a smooth muscle relaxant and improves FEV₁ by 10%; dosage is 25–75 mg/kg IV over 20 minutes.
- (7) Leukotriene modifiers are potent inflammatory mediators that play an important role in outpatient therapy but have not yet been shown to have a role in the acute exacerbation of asthma. Their bronchodilating effects are additive to those of β_2 -agonists, but further research is needed to investigate their role in acute asthma management.
- (8) Heliox (a mixture of helium and oxygen) should be considered for children with severe exacerbations and/or impending respiratory failure. By virtue of its lower density than that of air, it reduces airway resistance and, in turn, the work of breathing. It can provide additional time for first-line agents to take effect and so may avert intubation in these patients.
- (9) Methylxanthines are no longer recommended for routine management of acute asthma.
- (10) Noninvasive positive-pressure ventilation (CPAP or BiPAP) may successfully avert the need for intubation.
- (11) Ketamine is the agent of choice for patients in respiratory failure who require intubation because of its bronchodilatory effects.
- (12) Ventilator settings for intubated asthmatics should reflect a strategy of “permissive hypercapnia” (controlled hypoventilation) to minimize airway pressures and reduce the potential for barotrauma; use a tidal volume of 5–8 mL/kg in these patients.

VI. PEDIATRIC INFECTIOUS DISEASES

A. The febrile child

1. Pathophysiology of fever production: exogenous agents (bacterial endotoxins, antigen-antibody complexes) → formation and release of endogenous pyrogens (interleukins, tumor necrosis factor) → prostaglandin synthesis → raises the body's thermostatic set point → chills → shivering, peripheral vasoconstriction, behavioral activities (cover with blanket, put on a coat, etc) → increase in body temperature
2. Heat loss occurs secondary to
 - a. Radiation (60%)
 - b. Evaporation (25%)
 - c. Convection (10%)
 - d. Conduction (5%)
3. Temperature assessment
 - a. A rectal temperature is the most accurate and is, therefore, the gold standard and preferred route in almost all infants <6 months old in whom an accurate temperature is indicated.

- b. Tympanic thermometry is inaccurate in children <6 months old (low sensitivity, variable reproducibility) and should not be used to exclude fever in young children.
- 4. Risk of occult serious bacterial infection (bacteremia, meningitis, urinary tract infection, pneumonia) varies inversely with age.
 - a. Neonates are at highest risk.
 - b. Infants <1 month old with a rectal temperature $\geq 100.4^{\circ}\text{F}$ (38°C) should have a septic evaluation (CBC with differential, urinalysis, blood and urine cultures, lumbar puncture, and fecal culture when diarrhea is present, \pm chest radiograph). Children are then treated presumptively with antibiotics and admitted.
 - c. In children 1–2 months old, a septic evaluation is generally indicated, although the management and disposition may be individualized.
 - d. In infants 2–3 months old, the evaluation may be individualized and the lumbar puncture omitted if
 - (1) The infant appears well and is behaving normally (based on hydration status, quality of cry, reaction to parents, social responses).
 - (2) CBC is normal.
 - (3) Follow-up in 24 hours can be assured.
 - (4) No antibiotics are being administered.
- 5. Although the incidence of bacteremia generally increases with the degree of fever, infants 2–3 months old can have serious infections in the absence of high temperatures; in addition, neonates may present with hypothermia (temperature $<36.5^{\circ}\text{C}$).
- 6. Fever (particularly high fever) associated with leukocytosis often signifies a serious illness.
 - a. Historically, incidence of bacteremia in children 3–24 months old with a temperature $\geq 39.5^{\circ}\text{C}$ (103.1°F) is 5%–6% but climbs to 12%–15% in the presence of a WBC count $>15,000/\text{mm}^3$. However, immunization has decreased these incidences.
 - b. In children with hyperpyrexia, ie, a temperature $>105.8^{\circ}\text{F}$ (41°C), the rate of serious illness (bacteremia, bacterial meningitis) is even greater.
- 7. **Fever in association with a low WBC count ($<5,000/\text{mm}^3$) can also indicate serious disease. This is a classic finding in meningococcemia, septic shock, and disseminated intravascular coagulation.**
- 8. Fever associated with a petechial rash also places a child at higher risk of life-threatening bacterial infection (frequently due to infection with *Neisseria meningitidis* or *Haemophilus influenzae* type B).
- 9. Poisonings associated with fever
 - a. Anticholinergics (atropine), antihistamines
 - b. Salicylates
 - c. Amphetamines
 - d. LSD, phencyclidine (PCP), cocaine
 - e. Phenothiazines
 - f. Tricyclics
- 10. Management of fever
 - a. Acetaminophen 15–20 mg/kg/dose every 4 hours is still the drug of choice.
 - b. Ibuprofen 5–10 mg/kg/dose every 6–8 hours should be given to those infants >6 months old who do not respond to an adequate dose of acetaminophen.

- c. Aspirin is also effective, but is associated with Reye syndrome and should, therefore, be avoided.
- d. A tepid water (not alcohol) sponge bath may also be helpful.

B. Bacteremia and sepsis

1. Occult bacteremia = fever and positive blood culture in a well-appearing child without a major focus of infection
 - a. Most common organisms
 - (1) *Streptococcus pneumoniae*
 - (2) *Neisseria meningitidis*
 - (3) *Haemophilus influenzae*
 - (4) *Staphylococcus aureus*
 - (5) Group A streptococci
 - (6) *Salmonella* spp
 - b. Although children 3–36 months old are at risk of developing occult bacteremia, those 6–24 months old are most commonly affected; children <6 months old have protective antibodies, whereas those >24 months old are more immunocompetent.
 - c. Since the introduction of the heptavalent pneumococcal conjugate vaccine in 2000 and the vaccine for *H influenzae*, the prevalence of bacteremia continues to decline (current risk ~1%–2%). This is resulting in a reconsideration of occult bacteremia evaluation and management.
 - d. Historically, the risk of occult bacteremia is increased in the presence of:
 - (1) WBC count $\geq 15,000/\text{mm}^3$
 - (2) Temperature $\geq 102.2^\circ\text{F}$ (39.4°C)
 - (3) Absolute band count $> 1,500$
 - (4) Presence of toxic granulations or vacuolization of PMN leukocytes on peripheral smear
 - e. Occult bacteremia is associated with a risk of progression to a major focus of infection (such as meningitis).
 - (1) The risk of progression varies with the infecting organism.
 - (2) It is much greater with *H influenzae* and *N meningitidis* than with *S pneumoniae*.
2. Systemic inflammatory response system (SIRS)/sepsis/severe sepsis/septic shock
 - a. Definitions
 - (1) SIRS
 - (a) Widespread inflammatory response secondary to infection or other physiologic stressor
 - (b) SIRS criteria (must have two or more)
 - i. Temperature $> 101^\circ\text{F}$ (38.5°C) or $< 96.8^\circ\text{F}$ (36°C)
 - ii. Tachycardia: heart rate $> 95\text{th}$ percentile of normal for age; for children < 1 year old, bradycardia (heart rate $< 10\text{th}$ percentile of normal for age) also a criterion
 - iii. Tachypnea: respiratory rate $> 95\text{th}$ percentile normal for age
 - iv. WBC count either increased or decreased for age (or $> 10\%$ immature neutrophils)
 - (2) Sepsis: SIRS in the presence of or as a result of suspected or proven infection

- (3) Severe sepsis: sepsis plus one of the following:
 - (a) Cardiovascular organ dysfunction
 - (b) ARDS
 - (c) Two or more organ dysfunctions
- (4) Septic shock: sepsis plus cardiovascular organ dysfunction
- b. Most common organisms
 - (1) Neonatal period (0–4 weeks)
 - (a) Group B streptococci
 - (b) *Escherichia coli*
 - (c) *Listeria monocytogenes*
 - (2) Infancy and early childhood
 - (a) *S pneumoniae* (declining due to vaccine)
 - (b) *N meningitidis*
 - (c) *S aureus* (methicillin susceptible and methicillin resistant)
 - (d) *H influenzae* (almost eliminated because of vaccination)
- c. Sick cell disease/trait is associated with an increased risk of sepsis, particularly from encapsulated organisms such as *S pneumoniae* (400-fold increased risk), *Neisseria*, and *H influenzae* (5-fold increased risk).
- d. Septic infants (<3 months old)
 - (1) Frequently hypothermic rather than febrile
 - (2) Feed poorly and often do not thrive
 - (3) Appear toxic

C. Meningitis

- 1. Generally occurs as a complication of primary bacteremia but may result from direct extension of a contiguous infection such as paranasal sinusitis, mastoiditis, or otitis media
- 2. Clinical presentation (age dependent)
 - a. Infants <3 months old
 - (1) Decreased oral intake, vomiting
 - (2) Irritability when being handled (paradoxical irritability)
 - (3) Lethargy
 - (4) Fever or hypothermia
 - (5) Seizures
 - (6) Bulging fontanelle (late finding)
 - b. After the first year of life
 - (1) Nausea and vomiting
 - (2) Photophobia
 - (3) Headache
 - (4) Fever
 - (5) Nuchal rigidity
 - (6) Altered sensorium
 - (7) Seizures

- (8) Kernig sign: with the hip flexed, passive extension of the knee produces pain
- (9) Brudzinski sign: involuntary flexion of the hips evoked by passive flexion of the neck

3. Etiology

- a. Neonates: Group B streptococci, *E coli*, *Listeria monocytogenes*
- b. Infants 1–2 months old: Group B streptococci, *S pneumoniae*, *Neisseria meningitidis*
- c. Children >2 months old: *S pneumoniae*, *N meningitidis*, and (very rarely now) *H influenzae* type B

4. Indications for noncontrast head CT before lumbar puncture

- a. Immunocompromised state
- b. History of CNS disease
- c. Seizure within 1 week of presentation
- d. Abnormal neurologic examination
 - (1) Altered sensorium
 - (2) Gaze palsy
 - (3) Focal neurologic deficits
 - (4) Abnormal speech

5. Management

- a. Antibiotic therapy should be administered without delay, optimally within 30 minutes of presentation to the emergency department. Regimens vary with the child's age and presumed pathogens. Initial dosages of the preferred agents are as follows:
 - (1) Neonates
 - (a) Ampicillin 100 mg/kg IV plus gentamicin 2.5 mg/kg IV, or
 - (b) Ampicillin 100 mg/kg IV plus cefotaxime 50 mg/kg IV
 - (2) Infants 1–2 months
 - (a) Ampicillin 100 mg/kg IV plus cefotaxime 50 mg/kg IV, or
 - (b) Ampicillin 100 mg/kg IV plus ceftriaxone 100 mg/kg IV
 - (3) Children >2 months
 - (a) Ceftriaxone 100 mg/kg IV, or
 - (b) Cefotaxime 50 mg/kg IV
 - (c) If gram-positive cocci are identified on Gram stain of the CSF and there is concern regarding the presence of cephalosporin and penicillin-resistant strains of *S pneumoniae* in the area, vancomycin 15 mg/kg should be added to the above regimens in this age group.
- b. Corticosteroid therapy
 - (1) Steroids neutralize the host response to bacterial cell lysis (because of their anti-inflammatory properties).
 - (2) When dexamethasone is administered early, it is controversial whether it effectively decreases the incidence of neurologic sequelae (eg, sensorineural hearing loss) in children with bacterial meningitis due to *H influenzae* or *S pneumoniae*.
 - (3) Dexamethasone (at 0.15 mg/kg) can be administered before antibiotic therapy in children ≥ 6 weeks old with suspected bacterial meningitis due to *H influenzae* or *S pneumoniae* (although there is no clear-cut benefit of steroids in pneumococcal meningitis).

- c. Antiviral therapy: acyclovir (10 mg/kg IV every 8 hours) should be administered if there is any clinical suspicion that the infant has herpes meningoencephalitis, eg, maternal herpes, bloody CSF.
- d. Indications for antimicrobial chemoprophylaxis for meningococcal disease
 - (1) All intimate contacts of patients
 - (2) Prehospital and hospital personnel with direct exposure to respiratory secretions
 - (3) All daycare/nursery school contacts of these children
- e. Regimens
 - (1) Rifampin
 - (a) Infants <1 month: 5 mg/kg orally every 12 hours × 2 days
 - (b) Children >1 month: 10 mg/kg orally every 12 hours × 2 days
 - (c) Adults: 600 mg orally every 12 hours × 2 days
 - (2) Ciprofloxacin 500 mg orally once
 - (3) Azithromycin 10 mg/kg orally once (maximal dose 500 mg)

D. Pneumonia

- 1. Epidemiology and pathophysiology
 - a. Incidence is greatest in children 6–12 months old and decreases with age.
 - b. Principal route of acquisition is aspiration of infectious particles into the lower respiratory tract.
- 2. Etiology
 - a. The principal organisms responsible for producing pneumonia in children vary with:
 - (1) Age
 - (2) Immunization status
 - (3) Comorbid conditions
 - (4) Daycare attendance
 - b. Most pneumonias in children (60%–90%) are caused by viruses; it is only in the newborn period that bacterial pathogens predominate.
 - (1) Respiratory syncytial virus is the most common viral agent.
 - (2) *S pneumoniae* remains the predominant bacterial pathogen in all age groups beyond the newborn period (although the 7-valent protein-polysaccharide pneumococcal conjugate vaccine is changing this).
 - (3) Introduction of the *H influenzae* B vaccine has resulted in a significant decline in the incidence of infection due to *H influenzae*.
 - c. Common causes of pneumonia in children by age (listed in decreasing order of frequency) are the following:
 - (1) Newborns 0–2 weeks old
 - (a) Group B streptococci
 - (b) *E coli*
 - (c) *Klebsiella pneumoniae*
 - (d) *Listeria monocytogenes*
 - (2) Infants 2 weeks to 3 months old
 - (a) Viruses (respiratory syncytial virus, parainfluenza, human metapneumovirus)
 - (b) *Chlamydia trachomatis*

- (c) *S pneumoniae*
- (d) *H influenzae*
- (e) *S aureus*
- (3) Children 3 months to 4 years old
 - (a) Viruses
 - (b) *S pneumoniae*
 - (c) *H influenzae* (non-type B where vaccine not prevalent)
 - (d) *Mycoplasma pneumoniae*
- (4) Children 5–18 years old
 - (a) *Mycoplasma pneumoniae*
 - (b) Viruses
 - (c) *S pneumoniae*
- 3. Clinical pictures associated with specific infecting organisms
 - a. Viral pneumonia: a child presents with a history of preceding upper respiratory infection symptoms (often with an exanthem), followed by gradual onset of lower respiratory tract symptoms and a low-grade fever.
 - b. *Bordetella pertussis*: an afebrile infant with a 1–2 week history of mild upper respiratory tract symptoms and cough now presents with a severe paroxysmal cough and inspiratory whoop that is frequently followed by post-tussive emesis.
 - c. *Mycoplasma pneumoniae*: familial spread of a respiratory tract infection over a long period of time in a school-aged child with a nonproductive hacking cough, inspiratory rales, and multiple-organ involvement (pharyngitis, bullous myringitis, otitis media).
 - d. *Chlamydia pneumoniae*: an infant 2–16 weeks old has a “staccato” cough, is tachypneic, and usually afebrile; there is often a history of conjunctivitis and nasal congestion.
 - e. Pneumococcal pneumonia: an older child with abrupt onset of high fever, cough, and tachypnea; chest radiograph often has focal findings.
 - f. Staphylococcal pneumonia: a child with radiographic findings of pneumothorax and empyema (or pyopneumothorax)
- 4. Diagnostic evaluation
 - a. Pulse oximetry (to check for hypoxia)
 - b. Chest radiograph (mainstay of diagnosis)
 - (1) Useful in confirming the diagnosis and identifying complications
 - (2) May provide a clue as to the underlying cause of the pneumonia. Although there is considerable variability, a diffuse interstitial pattern suggests a viral or chlamydial infection, while alveolar infiltrates in a lobar distribution are more consistent with a bacterial infection. Pyopneumothorax suggests staphylococcal pneumonia.
 - c. CBC (nonspecific)
 - (1) High WBC count with a left shift suggests bacterial pneumonia.
 - (2) Lymphocytosis is common with viral, chlamydial, and pertussis pneumonia; a WBC count of 15,000–40,000/mm³ with a marked lymphocytosis is characteristic of pertussis.
 - (3) Normal WBC count and differential is typical of mycoplasmal pneumonia.
 - (4) WBC count <5,000/mm³ may indicate a poor prognosis.
 - (5) Eosinophilia suggests chlamydial pneumonia.

- d. Blood cultures should be ordered in all admitted, toxic-appearing, febrile children in whom bacterial pneumonia is suspected.
 - e. Increased cold agglutinins suggest mycoplasmal pneumonia.
5. Admission criteria
- a. Hypoxia (oxygen saturation $\leq 92\%$ – 93% at sea level)
 - b. Toxicity
 - c. Poor feeding/dehydration
 - d. Respiratory distress
 - e. Age <3 months old
 - f. Apnea in infants
 - g. Underlying medical problems, immunodeficiency
 - h. Presence of complications (pneumatocele, pleural effusion, empyema, pneumothorax)
 - i. Unresponsive to outpatient therapy
 - j. Unreliable caregiver
6. Management
- a. Viral pneumonia will resolve without specific antibiotic therapy and may be treated with supportive measures alone when the diagnosis is clear.
 - b. Bacterial pneumonia necessitates specific antimicrobial therapy that is based on the child's age and the likely pathogens seen in that age group. Because it is difficult to exclude bacterial disease with certainty on initial presentation, antibiotics are usually prescribed if the clinical picture suggests a treatable etiology.
 - c. Antibiotic therapy for bacterial pneumonia
 - (1) Newborns <4 weeks old
 - (a) Inpatient
 - i. Ampicillin and gentamicin or
 - ii. Ampicillin and cefotaxime
 - (b) Outpatient therapy is not recommended.
 - (2) Infants 1–3 months old
 - (a) Inpatient
 - i. Ampicillin and a third-generation cephalosporin
 - ii. Erythromycin/azithromycin should be added to the above regimen when infection with *Chlamydia trachomatis* or *Bordetella pertussis* is suspected.
 - (b) Outpatient therapy is not recommended.
 - (3) Children 3 months to 5 years old
 - (a) Inpatient
 - i. A second- or third-generation cephalosporin
 - ii. Erythromycin/azithromycin should be added to the above regimen if infection with *C trachomatis* or *Mycoplasma pneumoniae* is suspected.
 - (b) Outpatient (choose one)
 - i. Amoxicillin
 - ii. Amoxicillin-clavulanate
 - iii. Erythromycin-sulfamethoxazole
 - iv. Clarithromycin

- v. Azithromycin
 - vi. Cefuroxime axetil
 - vii. Cefixime
 - viii. Cefaclor
 - ix. IM ceftriaxone as a one-time dose followed by an oral second- or third-generation cephalosporin should be considered for children with signs of bacteremia.
- (4) Children 5–18 years old
- (a) Inpatient
 - i. A second- or third-generation cephalosporin
 - ii. Erythromycin/azithromycin should be added to the above regimen if infection with *C trachomatis* or *M pneumoniae* is suspected.
 - (b) Outpatient (choose one)
 - i. Erythromycin
 - ii. Clarithromycin
 - iii. Azithromycin
 - iv. Erythromycin/sulfamethoxazole
- (5) Children with severe disease or requiring ICU care: cefotaxime, azithromycin, and vancomycin
- d. Follow-up in 24–48 hours should be arranged.
- e. If a young child presents with persistent pneumonia (or recurrent pneumonia in the same location), the possibility of a missed foreign body aspiration or some other underlying disease process should be considered.

E. Pertussis (whooping cough)

1. Etiology
 - a. *Bordetella pertussis* (a gram-negative coccobacillus) is a highly contagious infection caused by inhalation of contaminated droplets.
 - b. Incubation period is 6–20 days with a mean of 7–10 days.
2. Epidemiology
 - a. Pertussis is seen primarily in nonimmunized or partially immunized children and adolescents but is also seen in adults because the immunization series does not guarantee life-time protection.
 - b. The highest morbidity rate is in children <1 year old; mortality is highest in the first month of life and then progressively declines.
3. Clinical presentation
 - a. Young children are most likely to have the classic three-stage illness.
 - (1) Catarrhal stage: mild fever, rhinorrhea, and conjunctivitis lasting up to 2 weeks
 - (2) Paroxysmal stage: unremitting paroxysmal coughing followed by a “whoop” (inspiratory stridor); as many as 40 episodes per day can occur for 2–4 weeks; vomiting in association with these paroxysms is common (although it may be absent in infants who present with decreased responsiveness, cyanosis, and bradycardia; there is a history of choking spells and apnea.)
 - (3) Convalescent stage: a residual cough that usually lasts 1–6 weeks but may persist for several months

- b. Older children and adults may be misdiagnosed as "chronic bronchitis," because they are less ill and frequently have only a persistent cough, rhinorrhea, and pharyngitis.
- 4. Diagnostic evaluation
 - a. WBC count typically ranges from 20,000–50,000/mm³ with a marked absolute lymphocytosis.
 - b. Chest radiograph may show peribronchial thickening or a "shaggy" heart border. If the primary illness is complicated by pneumonia, infiltrates may be seen.
 - c. The organism can be cultured on Bordet-Gengou medium using nasopharyngeal or sputum specimens. It can also be detected with fluorescent antibody staining of a nasopharyngeal swab specimen; a polymerase chain reaction test may be available in some centers.
- 5. Management
 - a. The antibiotic of choice is erythromycin 40–50 mg/kg/day orally in divided doses × 14 days (maximal dosage 2 g/day), or azithromycin 10 mg/kg/day on day 1 followed by 5 mg/kg/day on days 2–5 (infants <6 months old receive 10 mg/kg/day for 5 days). Erythromycin is not recommended for infants <1 month; azithromycin is the agent of choice.
 - b. Hospital admission with isolation measures is indicated for children <6 months old secondary to higher morbidity and mortality, and for older children with hypoxia and cyanosis during coughing spells or apnea. Pulmonary support with supplemental oxygen and postural drainage is important.
 - c. Prophylaxis with erythromycin for 10–14 days should be considered for household and other close contacts.
- 6. Potential complications
 - a. Pulmonary
 - (1) Pneumonia
 - (2) Pneumothorax
 - (3) Crepitus from subcutaneous or mediastinal emphysema
 - (4) Hypoxia
 - (5) Apnea
 - b. Neurologic
 - (1) Seizures
 - (2) Encephalitis
 - (3) Hypoxic encephalopathy
- F. Otitis media (see also pages 138–139)
 - 1. Pathophysiology
 - a. Obstruction or abnormal patency of the eustachian tube is the principal factor in the development of acute otitis media. Obstruction is frequently the result of inflammation from an antecedent or ongoing viral infection, and when it occurs in the presence of bacterial colonization of the nasopharynx, the stage is set for the development of middle ear infection.
 - b. Obstruction of the eustachian tube → negative pressure in the middle ear and development of a sterile effusion → aspiration of nasopharyngeal secretions in the middle ear → acute otitis media
 - c. Infants have short, more horizontal (and more easily collapsible) eustachian tubes than older children. These anatomic differences predispose infants to eustachian tube

obstruction and reflux of oropharyngeal secretions, thereby placing them at greater risk of development of acute otitis media.

2. Etiology

- a. *Streptococcus pneumoniae* (30%–40%)
- b. *Moraxella catarrhalis* (25%)
- c. Nontypeable *Haemophilus influenzae* (20%)
- d. *Streptococcus pyogenes* group A (3%)
- e. *Staphylococcus aureus*, group B streptococci, and gram-negative enteric bacilli (may be seen as pathogens in the neonatal period but are otherwise uncommon)
- f. Viral infection is probably the most common cause.

3. Clinical presentation

- a. The most reliable sign of acute otitis media is decreased mobility of the tympanic membrane on pneumatic otoscopy.
- b. Other findings include abnormal erythema, distortion or bulging of the tympanic membrane, or loss of bony landmarks.

4. Selection of antibiotics

- a. Some clinicians recommend withholding antibiotics in older children with mild signs and symptoms for 2–3 days when a virus is the suspected agent. Because most of these infections are self-limited, this reduces overtreatment and development of resistance.
- b. Amoxicillin 80–90 mg/kg/day (divided bid) is still the drug of choice.
- c. If β -lactamase-producing *H influenzae* and *M catarrhalis* is suspected or documented, amoxicillin-clavulanate or cefuroxime is preferred.
- d. Treatment failure with use of the above agents for 3 days necessitates the use of one of the following broad-spectrum antibiotics:
 - (1) Amoxicillin-clavulanate 40 mg/kg/day divided bid
 - (2) Azithromycin suspension 10 mg/kg as a single dose on day 1, then 5 mg/kg on days 2–5
 - (3) Cefuroxime axetil 30 mg/kg/day divided bid
 - (4) Cefpodoxime proxetil 10 mg/kg/day divided bid
 - (5) Cefdinir 14 mg/kg/day (can be divided bid) or clarithromycin 15 mg/kg/day divided bid
 - (6) Ceftriaxone 50 mg/kg in a single IM dose is particularly useful if vomiting, compliance, or follow-up is a problem.

Table 28: Recommended Antibiotics for Treatment of Otitis

Initial Antibiotic Treatment		Failure of Initial Antibiotic Treatment	
First-line	Alternative (penicillin-allergy)	First-line	Alternative
Amoxicillin	Cefdinir or cefuroxime	Amoxicillin-clavulanate	Ceftriaxone, clindamycin, with or without third-generation cephalosporin
Amoxicillin-clavulanate	Cefpodoxime or ceftriaxone	Ceftriaxone	Clindamycin plus third-generation cephalosporin Tympanocentesis Consult specialist

5. The possibility of systemic infection should be considered in infants with acute otitis media who have a fever and appear toxic (particularly those <2 months old). These infants require a septic evaluation and are often admitted for broad-spectrum parenteral antibiotic therapy. The incidence of infections due to coliforms, group B streptococci, and *S aureus* is higher in these infants. Coverage with ampicillin plus gentamicin, or ampicillin plus cefotaxime is highly recommended.
 6. Most children will be asymptomatic and afebrile within 2–3 days of the start of appropriate antimicrobial therapy; a lack of response is an indication for reevaluation and a possible change in antibiotic agents.
 7. Complications of acute otitis media
 - a. Extracranial
 - (1) Hearing loss (one of the most common complications)
 - (2) Tympanic membrane perforation (one of the most common complications)
 - (3) Tympanosclerosis
 - (4) Chronic suppurative otitis media
 - (5) Cholesteatoma
 - (6) Ossicular discontinuity and fixation
 - (7) Mastoiditis (clinical clues: tenderness over the mastoid process plus outward and downward displacement of the pinna)
 - (8) Labyrinthitis
 - (9) Facial nerve (cranial nerve VII) paralysis
 - b. Intracranial
 - (1) Meningitis (one of the most common complications)
 - (2) Lateral sinus thrombosis
 - (3) Encephalitis
 - (4) Abscess (brain, extradural)
 - (5) Subdural empyema
- G. Urinary tract infection (UTI)
1. Pathophysiology
 - a. In infants <3 months old, the incidence of UTI is 2–3 times greater in males than in females; uncircumcised males are more commonly affected. Thereafter, the incidence of UTI in males declines significantly, while the incidence in females increases and exceeds that in males.
 - b. Infection most commonly results from colonization of the urethral meatus with perineal flora; however, in young infants it may occur secondary to bacteremia.
 - c. Nearly 50% of children <1 year old with a UTI have vesicoureteral reflux or a structural abnormality of the urinary tract.
 - d. Associated sepsis is common in infants 1–3 months old with UTI (incidence of 30%).
 2. Etiology: usually coliforms, with *E coli* being the most common
 3. Clinical presentation of a neonate
 - a. History of fever, irritability, vomiting, diarrhea, poor feeding, or failure to thrive
 - b. On physical examination, the infant may appear jaundiced or septic.

4. Clinical presentation of a child 2–5 years old
 - a. Fever may be the only complaint (with a normal physical examination); may have a lower UTI (cystitis).
 - b. Dysuria and frequency may be conspicuously absent.
 - c. Occasionally, there is a history of enuresis or incontinence.
 - d. Lower abdominal pain (with little or no tenderness) should suggest the possibility of a UTI (upper and lower tract).
5. Male children with a UTI should have an examination of the external genitalia to exclude the following:
 - a. Phimosis: the distal foreskin cannot be retracted.
 - b. Paraphimosis: the foreskin is swollen and retracted.
 - c. Urethritis
 - d. Meatal stenosis (most common cause of dysuria in this age group (2–5 years old))
6. Female children with symptoms of a UTI should be examined to exclude vulvovaginitis.
7. Diagnostic evaluation
 - a. Urinalysis
 - (1) Specimen collection
 - (a) In infants and young children, specimens should be obtained by bladder catheterization or suprapubic aspiration; bag specimens are often contaminated and are, therefore, unreliable.
 - (b) In older children who are toilet-trained, an appropriately collected midstream clean-catch specimen is adequate.
 - (2) Pyuria (>10 WBCs per high-power field of spun urine) is suggestive of UTI but not diagnostic (also occurs in association with other inflammatory processes).
 - (a) Renal causes
 - i. Pyelonephritis (WBC casts on urinalysis)
 - ii. Perinephric abscess
 - iii. Acute glomerulonephritis (RBCs, RBC casts, and proteinuria)
 - (b) Extrarenal causes
 - i. Appendicitis
 - ii. Vulvovaginitis
 - iii. Vaginal foreign body
 - iv. Gastroenteritis
 - v. Dehydration
 - vi. Sexually transmitted infections
 - (3) Bacteriuria is a more reliable finding; there is a 95% probability of infection with 100,000 colony-forming units/mL. Presence of any bacteria on Gram stain of a noncentrifuged specimen is the best combination of high sensitivity and a low false-positive rate.
 - (4) Nitrite test: detects nitrites produced by the reduction of dietary nitrates by urinary gram-negative bacteria (especially *E coli*, *Klebsiella* spp, and *Proteus* spp)
 - (a) Positive test is virtually diagnostic for UTI (high specificity).
 - (b) False-negative results are common (low sensitivity).

- (5) Leukocyte esterase test
 - (a) Detects esterases released from degraded leukocytes (an indirect test for WBCs)
 - (b) Poor sensitivity and specificity for UTI
- b. Urine culture confirms the diagnosis and should be sent for:
 - (1) Females <3 years old
 - (2) Males <1 year old (even if the urinalysis is negative) and those who are uncircumcised
- 8. Management: antibiotic therapy
 - a. Aimed at the coliforms and should be started in the symptomatic child with pyuria or bacteriuria
 - b. Adolescents with uncomplicated cystitis may be treated with antibiotics for 3–5 days. Children with lower tract infections, however, should be treated for 7–10 days.
 - c. Acceptable alternatives include amoxicillin, sulfisoxazole, TMP-SMX, and cephalexin.
 - d. Antibiotic selection should reflect resistance patterns in your community.
 - e. Children with evidence of pyelonephritis (particularly those <3 months old and those who appear toxic) are generally hospitalized for parenteral antibiotic therapy (ampicillin plus gentamicin) for 10 days.
- 9. Radiographic evaluation
 - a. Initial screening test of choice is ultrasonography.
 - b. Should be performed in the following children:
 - (1) Neonates and infants of both sexes
 - (2) All males regardless of age
 - (3) Females 1–5 years old
 - (4) All children with a recurrent UTI
- H. Kawasaki disease (mucocutaneous lymph node syndrome)
 - 1. Definition: an acute multisystem vasculitis of small and medium-sized arteries with a particular predilection for the coronary arteries
 - 2. Epidemiology
 - a. Infants and children are affected most commonly; 80% of cases occur in children <5 years old, with peak incidence in those 1–2 years old.
 - b. All racial groups are affected, but children of Asian descent are at greatest risk.
 - c. Male to female ratio is 1.5 to 1.
 - d. Incidence is greatest in the winter and spring.
 - 3. Diagnostic criteria
 - a. Fever ≥ 5 days (usually high) plus
 - b. Presence of four of the following five conditions:
 - (1) Bilateral nonsuppurative conjunctivitis
 - (2) Changes of the lips and oral mucosa
 - (a) Erythematous or fissured lips
 - (b) Strawberry tongue
 - (c) Injected oropharynx

- (3) **Extremity features**
 - (a) **Palmar and plantar erythema**
 - (b) **Indurative edema of hands and feet**
 - (c) **Periungual desquamation**
- (4) **Polymorphous rash, which is most widespread on the trunk and proximal extremities with particular prominence in the perineal area**
- (5) **Cervical lymphadenopathy (at least one node >1.5 cm) that is usually unilateral occurs in 50%–75% of cases.**
- c. **Findings cannot be attributed to another disease process.**
- 4. **Incomplete (atypical) Kawasaki disease**
 - a. **Diagnostic criteria include fever for >5 days plus**
 - (1) **Two or three of above characteristics**
 - (2) **C-reactive protein >3 mg/dL, and erythrocyte sedimentation rate >40 mm/hr**
 - b. **Management is determined by echocardiogram findings.**
- 5. **Disease course is divided into three phases.**
 - a. **Acute phase (days 1–11)**
 - (1) **Characterized by fever and most of the other diagnostic features described above**
 - (2) **Myocarditis is the most common cause of death during this time.**
 - b. **Subacute phase (days 11–20)**
 - (1) **Characterized by gradual resolution of fever, rash, and adenopathy, as well as the development of thrombocytosis and periungual desquamation**
 - (2) **Risk of developing coronary artery thrombosis is greatest at this time.**
 - (3) **Causes of death include myocardial infarction, aneurysm rupture, and myocarditis.**
 - c. **Convalescent phase (days 21–60)**
 - (1) **Begins when all signs and symptoms have disappeared and continues until the erythrocyte sedimentation rate and platelet count have normalized.**
 - (2) **Death may occur from myocardial infarction secondary to thrombosis.**
- 6. **Diagnostic evaluation**
 - a. **ECG**
 - b. **Echocardiography (to assess cardiac status)**
 - c. **Chest radiograph**
 - d. **CBC with platelet count**
 - (1) **Moderate leukocytosis with left shift (acute phase)**
 - (2) **Mild normochromic, normocytic anemia**
 - (3) **Increased platelet count (subacute phase)**
 - e. **Erythrocyte sedimentation rate increased**
 - f. **C-reactive protein increased**
 - g. **Serum transaminases mildly increased**
 - h. **Urinalysis**
 - (1) **Sterile pyuria (WBCs without bacteria)**
 - (2) **Proteinuria**
 - i. **Studies to exclude other diagnoses (streptococcal culture, blood culture, ASO titer, etc)**

7. Management
 - a. Hospital admission (unit chosen depends on the extent of cardiac findings)
 - b. Early consultation with a pediatric cardiologist
 - c. IV immunoglobulin
 - (1) Dosage is 2 g/kg over 10–12 hours.
 - (2) When administered early in the illness (within 10 days of onset), it decreases systemic inflammation and the prevalence of coronary artery aneurysms.
 - d. Oral aspirin
 - (1) Anti-inflammatory dosage (80–100 mg/kg/day divided qid) during the acute phase
 - (2) Antithrombotic dosage (3–5 mg/kg/day as a single dose) during the subacute and convalescent phases
 - e. Steroids are not indicated and may increase the risk of coronary aneurysm development.
8. Complications
 - a. Cardiovascular (most important)
 - (1) A child's prognosis and outcome are primarily determined by the cardiac complications of Kawasaki disease.
 - (2) Coronary artery aneurysms and ectasia develop in 20%–30% of untreated children; those <1 year old are at greatest risk.
 - (3) With early treatment, the incidence of coronary artery aneurysm is reduced to 2%–4%.
 - b. Hydrops of the gallbladder occurs in 3% of patients and is a self-limiting complication.
 - c. Meningoencephalitis
9. Mortality and morbidity
 - a. Kawasaki disease is the most frequent cause of acquired pediatric cardiac disease in the United States.
 - b. Early and more effective treatment (IV immunoglobulin) has reduced the death rate to <1%.
 - c. Death is usually due to cardiac complications and generally occurs in the subacute and convalescent phases.
 - d. Myocardial infarction secondary to thrombotic occlusion in an aneurysmal or stenotic artery is the most common cause of death. Other less frequent causes include dysrhythmias, aneurysm rupture, and myocarditis.

VII. PEDIATRIC SEIZURES

A. Most common types

1. Simple febrile seizures
 - a. Affect 3%–5% of children
 - b. Most common seizure disorder of childhood; a family history of similar seizures is common.
 - c. Occur in children 6 months to 5 years old
 - d. Defined as an event of infancy or childhood that occurs in association with fever but without evidence of intracranial infection or a specific cause

- e. Clinical presentation
 - (1) Usually associated with a rapidly rising fever
 - (2) Last <10–15 minutes
 - (3) Generalized (not focal)
 - (4) Occur as a single event in a 24-hour time period
 - (5) Focal postictal neurologic deficits are absent.
 - f. Diagnostic evaluation
 - (1) Evaluation should be directed at identifying the source of infection.
 - (2) Routine laboratory or radiographic studies have no role.
 - (3) A lumbar puncture (to exclude intracranial infection) is indicated in children with meningeal signs (eg, nuchal rigidity, or Kernig or Brudzinski sign) and should be considered in those already on antibiotics to exclude the possibility of a partially treated meningitis. Other possible indications are in children with recurrent, partial, or prolonged seizures or in those with a prolonged postictal period.
 - g. Treatment is directed at lowering the fever and identifying any underlying infection.
 - h. Seizures recur in 30%–40% of children; more likely when the first febrile seizure occurs before 1 year old.
 - i. Prophylaxis with phenobarbital is not routinely recommended; although it is the drug of choice, it has not been shown to be of significant benefit and interferes with cognitive functioning.
2. Generalized tonic-clonic seizures
- a. Clinical presentation
 - (1) Both hemispheres are involved.
 - (2) Motor involvement is bilateral.
 - (3) Consciousness is impaired.
 - (4) Patients are confused and lethargic during the postictal period.
 - b. Diagnostic evaluation
 - (1) Includes a directed history (description of the seizure and surrounding events, pertinent past medical history) and a complete physical examination
 - (2) No role for routine laboratory or radiographic evaluation unless suggested by the history and physical examination.
 - (3) Tests ordered should reflect the child's age and suspected cause of the seizure as suggested by the history and physical examination.
 - (a) Electrolytes
 - (b) Toxicology screening
 - (c) Anticonvulsant medication serum levels (if applicable)
 - (4) Indications for a CT scan of the head
 - (a) Focal neurologic deficits
 - (b) Signs of increased intracranial pressure
 - (c) Infants <12 months old (possible abuse)
 - (d) Children with ventriculoperitoneal shunts
 - (e) Suspected head trauma
 - (5) MRI
 - (a) Preferred neuroimaging study

- (b) More sensitive than CT for detecting structural abnormalities
- (c) No radiation exposure
- (6) Lumbar puncture should be performed if there is a concern for meningitis or encephalitis.
- (7) Electroencephalogram shows generalized epileptic activity.
- c. Management
 - (1) Benzodiazepines will terminate >80% of seizures. Use one of the following:
 - (a) Lorazepam 0.1 mg/kg IV is the preferred agent; it has a long half-life and the least effect on respiratory depression.
 - (b) Diazepam 0.2–0.3 mg/kg IV may be used if lorazepam is unavailable; it has a much shorter half-life and produces a greater respiratory depression.
 - (c) Midazolam 0.2 mg/kg IM or intranasal should be considered when IV access cannot be rapidly attained; it works as rapidly by this route as by the IV route but is very short acting.
 - (d) Diazepam 0.5 mg/kg may also be given rectally if IV access cannot be attained and midazolam is unavailable; when administered via this route, its onset of action is ~5 minutes.
 - (2) If seizure activity persists despite administration of a benzodiazepine, fosphenytoin (15–20 phenytoin equivalents [PE]/kg diluted in normal saline and infused at a rate ≤ 50 mg/min) or phenobarbital (15–20 mg/kg infused at a rate of 25 mg/min) should be administered.
 - (3) Children presenting with their first afebrile, unprovoked seizure who make an uneventful recovery need not be treated with a long-term anticonvulsant unless they presented in status epilepticus. The rate of seizure recurrence in these children is quite variable; risk of recurrence within 1 year is ~25%; children with an underlying neurologic disease have a much higher recurrence rate (16%–62%), and the adverse effects associated with anticonvulsant therapy are significant. Therefore, it is recommended that anticonvulsant therapy be withheld until a second seizure occurs in these patients.
 - (4) Most breakthrough seizures are a result of subtherapeutic drug levels or noncompliance.
 - (5) Referral is generally indicated for ongoing monitoring.
- 3. Infantile spasms
 - a. Definition: convulsive disorders of infants and young children with onset between 3 and 9 months old and accompanied by developmental regression
 - b. Clinical presentation
 - (1) Spasms last only a split second and are associated with flexion or extension of the head and trunk.
 - (2) Spasms may occur as a single event or in groups of 5–20 at a time.
 - c. Electroencephalogram is almost always abnormal with hypsarrhythmia (a slow irregular rhythm) being present in 50% of cases.
 - d. Management includes admission and a neurologic consult.

B. Differential diagnosis

- 1. Syncope
- 2. Pseudoseizure

3. Breath-holding spells
4. Movement disorders (eg, tics)
5. Night terrors
6. Cardiac dysrhythmias
7. Complicated migraines

C. Etiology

1. Infectious
 - a. Meningitis/encephalitis
 - b. Herpes simplex virus
 - c. Bacterial meningitis
 - d. Parasitic encephalitis
2. Metabolic
 - a. Hypoglycemia
 - b. Hypo- or hypernatremia
 - c. Hypocalcemia
 - d. Hypoxia
 - e. Pyridoxine deficiency
3. Inborn errors of metabolism
4. Traumatic
5. Neoplastic
6. Drug-related (intoxication or withdrawal)
7. Child abuse (eg, shaken baby syndrome)
8. Miscellaneous
 - a. Congenital malformations
 - b. Ventriculoperitoneal shunt malfunction
 - c. Idiopathic

D. Adverse effects of medications used in management of pediatric seizures

1. Phenytoin: gum hyperplasia, hirsutism, and drug rashes (Stevens-Johnson syndrome)
2. Phenobarbital: lethargy, hyperactivity, and behavioral disorders
3. Carbamazepine: neutropenia, rashes, hepatic dysfunction, and cardiac toxicity
4. Valproic acid: thrombocytopenia, pancreatitis, hepatic failure, and a bleeding diathesis when used in conjunction with aspirin

VIII. CEREBROSPINAL FLUID SHUNTS

A. Shunt systems

1. Generally consist of a ventricular catheter, a valve device, and distal tubing
2. Ventriculoperitoneal shunts are the most common type, accounting for >90% of the shunts currently in use.
3. Ventriculoperitoneal shunts allow CSF to be diverted around obstructed areas in the subarachnoid space or ventricles.

4. Conditions that result in hydrocephalus and require CSF shunt placement include:
 - a. Congenital stenosis of the aqueduct of Sylvius
 - b. Acquired aqueductal stenosis or scarring secondary to infection
 - c. Malignancy or hemorrhage
 - d. Dandy-Walker malformations
 - e. Arnold-Chiari syndrome
5. Shunt failure is common; it can result from:
 - a. Intracranial infection or blockage
 - b. Valve malfunction
 - c. Distal tubal obstruction or disruption
 - d. Pseudocyst formation or disruption

B. Complications

1. Shunt malfunction
 - a. Obstruction
 - (1) Most common cause of shunt malfunction
 - (2) Results from blockage to flow anywhere along the shunt system and may be caused by kinking, thrombosis, disconnection, migration, or infection of the catheter.
 - (3) Patients present with signs of increased intracranial pressure.
 - (a) Headache with mental status changes and associated nausea and vomiting are the classic symptoms. Other clues include:
 - i. Seizures while on therapeutic medication doses
 - ii. Poor sucking or feeding
 - iii. Paradoxical crying
 - (b) Findings on physical examination
 - i. Upward gaze palsy ("sunsetting" or "sundowning")
 - ii. Diplopia
 - iii. Bulging fontanelle
 - iv. Enlarging head
 - v. Cushing triad of bradycardia, hypertension, and respiratory ataxia (preherniation syndrome)
 - (4) If left untreated, it can result in herniation.
 - b. Slit ventricle syndrome
 - (1) Results from chronic overdrainage of CSF with collapse of the ventricles and transient obstruction of CSF flow; overdrainage → collapse of the ventricles and catheter obstruction → increased intracranial pressure → reexpansion of the ventricular system and relief of catheter obstruction
 - (2) Symptoms are those of an intermittently increased intracranial pressure.
 - (a) Episodic minor headache associated with reduced performance and intervening asymptomatic periods are characteristic.
 - (b) Severe headache, nausea, vomiting, and bradycardia progressing to obtundation may occur with more severe presentations.
 - (3) CT reveals small ventricles.

- (4) Placement of the patient in Trendelenburg position frequently relieves these symptoms.
 - (5) Definitive treatment involves placement of an antisiphon device.
 - c. Evaluation of shunt malfunction
 - (1) Locate the shunt and trace its course, looking for evidence of infection and palpating for disconnections.
 - (2) Obtain a shunt survey (plain radiographs of the entire shunt system) to exclude kinks and breaks in the catheter.
 - (3) Compress the subcutaneous shunt reservoir and pumping chamber to evaluate ease of pump compression/refill (may be helpful but should not be used to exclude shunt dysfunction).
 - (a) When functioning normally, the chamber refills in <3 seconds.
 - (b) Inability to depress the chamber (high resistance) is consistent with a distal obstruction.
 - (c) Delayed or absent refilling of the chamber signifies proximal obstruction or overdrainage of the system.
 - (d) An audible clicking noise with compression of the chamber may signal an incompetent valve.
 - (4) Determine ventricular size via CT scanning, and compare the results with those of prior studies to see if there has been a change. In infants with open fontanelles, ventricle size can also be assessed by ultrasound. Again, comparative studies are necessary.
 - (a) Enlarged ventricles indicate obstruction.
 - (b) Smaller or slit ventricles are consistent with overdrainage.
 - (5) In difficult cases, radionuclide shunt clearance scans or MRI may be helpful.
 - (6) Management
 - (a) Obtain urgent neurosurgical consult.
 - (b) If seizures are secondary to increased intracranial pressure, manage as appropriate.
2. Shunt infections
- a. 60% of the mortality that occurs in association with shunts is due to shunt infections.
 - b. Most infections (75%) occur within the first 2 months after shunt placement and are caused by *S epidermidis* and *S aureus*.
 - c. Resulting shunt obstruction that leads to increased intracranial pressure is common.
 - d. Clinical presentation
 - (1) Fever is the most common symptom of shunt infection.
 - (2) Other findings
 - (a) Nausea, vomiting, headache, and feeding problems
 - (b) Meningismus
 - (c) A subtle change in behavior as detected by the parents
 - (3) Signs of increased intracranial pressure
 - e. Diagnostic evaluation
 - (1) Examine the course of the shunt looking for evidence of infection; although this is very specific for shunt infection, it is not sensitive.

- (2) Obtain laboratory studies.
 - (a) CBC with differential (may be normal)
 - (b) Blood culture
 - (c) CSF from the shunt (consult with neurosurgeon) for evaluation of samples for cell count and differential, glucose, Gram stain, protein, and culture and sensitivity
- f. Management
 - (1) Obtain urgent neurosurgical consult, and admit these patients to the hospital for antibiotic therapy (IV and/or intraventricular) and externalization of the shunt.
 - (2) Shunt removal and replacement is required in 50% of patients.

IX. PEDIATRIC GASTROINTESTINAL/GENITOURINARY EMERGENCIES

A. Abdominal conditions associated with rectal bleeding

1. Infectious colitis: infection with the organisms listed below can result in bloody diarrhea.
 - a. *Shigella*
 - b. *Salmonella*
 - c. Enteroinvasive *E coli*
 - d. *Campylobacter jejuni*
 - e. *Yersinia enterocolitica*
 - f. *Clostridium difficile*
 - g. *Entamoeba histolytica*
 - h. Enterohemorrhagic *E coli* serotype O157:H7
2. Painless rectal bleeding
 - a. Anal fissure (most common cause in infants)
 - b. Swallowed maternal blood
 - (1) Occurs in infants <2 months old
 - (2) Can be distinguished from fetal blood by the Apt test
 - c. Meckel's diverticulum
 - d. Infectious gastroenteritis
 - e. Juvenile polyps
3. Meckel's diverticulum can produce a variety of signs and symptoms.
 - a. It is a true diverticulum (all layers of the bowel wall are present) and can contain a variety of heterotopic tissue, of which gastric mucosa is the most common.
 - b. Painless rectal bleeding (which can be massive) is the most common presentation of this congenital anomaly in children <5 years old; ectopic gastric mucosa within the diverticulum → ulcer formation (in either the diverticulum or the nearby ileum) → bleeding
 - c. Meckel's isotope scanning is the most specific diagnostic study and should be ordered if this diagnosis is suspected. Although false-negative results can occasionally occur,

this is uncommon when modern techniques are used unless the amount of ectopic gastric mucosa is very small.

- d. Meckel's diverticulum can also be the focus about which volvulus, intussusception of the small bowel, or an internal hernia develops, all of which cause signs of intestinal obstruction.
 - e. When the diverticulum becomes inflamed, it can result in symptoms mimicking those of appendicitis.
- 4. Midgut volvulus**
- a. Most commonly a complication of congenital malrotation of the bowel → inadequate attachment to the mesentery
 - b. Generally occurs in infants <1 year old, with most cases presenting within the first month of life
 - c. Clinical presentation
 - (1) Bilious vomiting, abdominal distention, tenderness, and a palpable mass
 - (2) Because the obstruction is high and decompressed by vomiting, abdominal distention may or may not be present.
 - (3) Rectal bleeding may occur within several hours and suggests bowel ischemia/necrosis.
 - d. Can produce gangrene of the midgut if not promptly treated
 - e. Diagnostic evaluation
 - (1) Plain radiographs are usually suggestive but may be deceptively normal in appearance. Presence of small bowel overlying the liver suggests an underlying malrotation. Gaseous distention of the stomach as well as dilated loops of small bowel with air-fluid levels suggest obstruction.
 - (2) Upper GI or ultrasound will confirm the diagnosis. A "corkscrew" or "apple core" sign is seen with the spiraling of the small intestine.
 - f. Management
 - (1) IV fluid resuscitation
 - (2) Nasogastric tube decompression
 - (3) Immediate surgical consult for emergent laparotomy
- 5. Intussusception: the most common cause of intestinal obstruction in children 3 months to 5 years old**
- a. Infants 6–12 months old are most commonly affected; male to female ratio is 4:1.
 - b. Ileocolic intussusception is the most common type.
 - c. Clinical presentation
 - (1) The classic triad of symptoms (present in only 21% of patients) consists of intermittent colicky abdominal pain, vomiting, and currant jelly feces.
 - (2) Unexplained altered mental status
 - (3) Lethargy can also be a prominent feature, particularly in infants <1 year old.
 - (4) Abdominal pain is usually the first symptom to develop; it lasts for a few minutes, during which the infant often screams and draws its legs up, and is followed by pain-free intervals of 10–20 minutes, during which the infant may appear happy and content or quiet and listless.
 - (5) Vomiting generally develops after a period of 6–12 hours and may become bilious.

- (6) Currant jelly feces are a late finding (develop >12 hours after the onset of pain) and occur in only 50% of patients; fecal guaiac, however, is frequently positive.
- (7) Examination may reveal a sausage-shaped mass in the right upper quadrant with absence of bowel sounds in the right lower quadrant.
- (8) Absence of currant jelly feces and/or an abdominal mass does not exclude the diagnosis and should not delay further evaluation.
- (9) Associated with Henoch-Schonlein purpura; an ultrasound should be obtained in patients complaining of abdominal pain or vomiting.
- d. Diagnostic evaluation
 - (1) Plain radiographs should be obtained first.
 - (a) Findings
 - i. An abdominal mass or filling defect in the right upper quadrant
 - ii. Signs of bowel obstruction
 - iii. Free intraperitoneal air
 - (b) Normal plain radiographs do not exclude the diagnosis.
 - (2) Ultrasound evaluation is a quick and sensitive modality to identify an intussusception. However, air insufflation and barium enema are the preferred diagnostic and treatment studies.
 - (a) Air-contrast is as effective as barium in reducing the intussusception and has replaced the barium enema as the study of choice in most major medical centers; successful reduction occurs in 60%–80% of cases.
 - (b) If barium enema is performed, the classic finding of a “coiled spring” appearance may be seen on the evacuation radiograph.
 - e. Operative intervention is required for patients in whom the intussusception cannot be reduced by air insufflation or barium enema.
 - f. Recurrence rate is 5%–10% after barium enema reduction and 2%–5% after surgical reduction, with most recurrences occurring in the first 24 hours after reduction.
- B. Abdominal/genitourinary conditions not associated with rectal bleeding (these are all surgical emergencies)
 - 1. Incarcerated hernia
 - a. Clinical presentation
 - (1) Common in children <1 year old
 - (2) Symptoms include vomiting and irritability accompanied by a scrotal or inguinal mass.
 - b. Differential diagnosis
 - (1) Hydrocele
 - (2) Undescended testes
 - (3) Testicular torsion
 - (4) Torsion of the appendix testis
 - (5) Inguinal lymphadenopathy or node abscess
 - c. Management
 - (1) Manual reduction (with or without sedation) followed by surgical referral on an outpatient basis to schedule elective repair
 - (2) If manual reduction is unsuccessful, obtain prompt surgical consultation for surgical reduction.

2. Intestinal obstruction
 - a. Clinical presentation: vomiting with abdominal pain and distention
 - b. Differential diagnosis
 - (1) Intestinal atresia/stenosis (newborn or infant)
 - (2) Meconium ileus (newborn)
 - (3) Incarcerated inguinal hernia
 - (4) Malrotation with or without volvulus
 - (5) Volvulus
 - (6) Imperforate anus
 - (7) Hirschsprung disease
 - c. Diagnostic evaluation: plain radiographs typically show dilated loops of bowel with air-fluid levels.
 - d. Management
 - (1) IV fluid resuscitation
 - (2) Nasogastric tube decompression
 - (3) Immediate surgical consult
3. Pyloric stenosis
 - a. **Occurs most commonly in males 2–6 weeks old (particularly firstborns); a positive family history is common, especially if the mother had it as an infant.**
 - b. Pathophysiology: smooth muscle hypertrophy of the pylorus → gastric outlet obstruction
 - c. Clinical presentation
 - (1) **Nonbilious projectile vomiting after feeding, after which the infant usually hungrily re-feeds**
 - (2) Evidence of dehydration and failure to thrive
 - (3) Prominent peristaltic waves (sometimes)
 - (4) Palpable olive-shaped mass (the hypertrophied pylorus) in the right upper quadrant (pathognomonic)
 - (5) Jaundice (~5% of cases)
 - d. Diagnostic evaluation
 - (1) When the “olive” can be palpated, the diagnosis is established and further diagnostic studies are unnecessary.
 - (2) When the “olive” cannot be palpated, ultrasound is the diagnostic study of choice; a hypoechogenic ring with a hyperdense center that resembles a “doughnut” or “bull’s-eye” can be seen.
 - (3) If the ultrasound is unavailable or negative, an upper GI series should be performed. Findings include the following:
 - (a) Delayed gastric emptying
 - (b) Indentation of the antrum by the “olive”
 - (c) Elongation of the pyloric channel (the “string” or “beak” sign)
 - (4) **Hypochloremic, hypokalemic metabolic alkalosis**

- e. Management
 - (1) Correct dehydration and electrolyte imbalance.
 - (2) Restrict oral feedings.
 - (3) Obtain early surgical consult; definitive treatment (once fluid and electrolyte balance have been restored) is pyloromyotomy.
- 4. Appendicitis
 - a. Epidemiology
 - (1) Most common nontraumatic surgical emergency in children
 - (2) Peak incidence 9–12 years old
 - (3) Rare in children <2 years old
 - b. Clinical presentation
 - (1) Evolution of symptoms: anorexia and periumbilical pain → vomiting → right lower quadrant pain that is worse when walking
 - (2) Right lower quadrant tenderness with signs of peritoneal irritability (guarding or rebound)
 - (3) Supportive findings: temperature is usually mildly increased.
 - c. Diagnostic evaluation
 - (1) WBC count is generally moderately increased with an increase in the number of PMNs and immature forms. Increased WBC count ($>10,000$ cells/mm³) is associated with the presence of appendicitis but has poor sensitivity and specificity and no clinical utility. Urinalysis may reveal sterile pyuria if the inflamed appendix is overlying the ureter.
 - (2) Chest radiograph with supine and upright abdominal films are helpful in excluding pneumonia presenting as abdominal pain; abdominal radiograph may demonstrate a fecalith (10% of cases), loss of psoas shadow, localized ileus in right lower quadrant.
 - (3) Ultrasound may be diagnostic but is highly operator dependent.
 - (4) CT scanning is being increasingly used to confirm the diagnosis.
 - d. Findings suggestive of perforation
 - (1) High temperature ($>102^{\circ}\text{F}$ [39°C])
 - (2) High WBC count ($>15,000/\text{mm}^3$)
 - (3) Symptoms >36 hours
 - e. Management
 - (1) IV fluid resuscitation
 - (2) Antibiotics (ampicillin, gentamicin, and metronidazole or clindamycin; piperacillin/tazobactam or cefoxitin; meropenem if perforation)
 - (3) Immediate surgical consult
- 5. Testicular torsion
 - a. General
 - (1) Annual incidence 4.5 in 100,000 males 1–25 years old
 - (2) Cannot be diagnosed by physical examination alone
 - (3) Salvage rate: classically 80%–100% within 6 hours of onset and $<10\%$ after 24 hours, but more recent literature suggests higher salvage rates despite later presentation.

- b. Clinical presentation
 - (1) Sudden onset scrotal pain with erythema and edema
 - (2) Nausea and vomiting
 - (3) Cremasteric reflex absent; transverse lie (although clinically neither has proved to be true)
 - (4) Prehn's sign: elevation of the testicle relieves pain from epididymitis but not torsion (unreliable).
- c. Diagnostic evaluation
 - (1) Color Doppler ultrasound or radionuclide testicular scan
 - (2) Surgical exploration
- d. Management
 - (1) Manual detorsion may be attempted as a temporary measure ("open the book" technique).
 - (2) Immediate urologic consult for surgical exploration and orchiopexy

X. CHILD ABUSE

A. Child neglect

- 1. Associated with failure to thrive
- 2. Clinical presentation (widely variable)
 - a. Behaviorally, the child:
 - (1) Is "wide-eyed" and "wary" and avoids eye contact
 - (2) Is difficult to console
 - (3) Demonstrates a preference for inanimate over animate objects
 - b. Physical findings typically include:
 - (1) Inadequate body weight for age
 - (2) Poor hygiene
 - (3) Increased muscle tone
 - (4) Chronic diaper rash
 - (5) Alopecia in association with a flattened occiput
- 3. Diagnostic evaluation and management
 - a. Obtain a skeletal survey to exclude associated physical abuse. Include AP and lateral views of the skull, chest, and spine, as well as an AP view of the pelvis and long bones.
 - b. Notify child protective services agency.
 - c. Admit to hospital for further evaluation; weight gain in the hospital setting is considered to be the sine qua non of failure to thrive due to child neglect.

B. Munchausen syndrome by proxy (Polle syndrome)

- 1. Definition: a form of child abuse in which a parent makes up or induces an illness in a child to gain attention or sympathy from healthcare providers and others

2. The illness is fabricated by one or a combination of three methods:
 - a. Lying
 - b. Simulation
 - c. Induction via medications or poisons (most dangerous method)
3. Typical characteristics of the parent/perpetrator
 - a. Biological mother
 - b. Medical background
 - c. Cooperative, concerned, and overly enthusiastic about performing additional studies
4. Clues to the diagnosis
 - a. Laboratory results do not "fit."
 - b. Signs and symptoms do not respond to appropriate medical management.
 - c. Signs and symptoms resolve when the child is separated from the perpetrator.
5. Management
 - a. Admit to hospital with continuous camera monitoring to assure safety.
 - b. Institute appropriate therapy.
 - c. Involve counseling and social services.

C. Sexual abuse

1. 20% of all girls and 9% of boys are sexually abused. The abuse has often occurred over a long period of time and, in 90% of cases, the perpetrator is well known to the child (eg, family member, relative).
2. Physical examination
 - a. Best performed with the child supine in the frog-leg position or prone in the knee-chest position; can be augmented by the use of a hand-held lens or colposcope
 - b. Suggestive findings
 - (1) Hymenal notches (concavities), scars, or tears
 - (2) Vaginal discharge
 - (3) Genital ulcers, warts, or vesicles
 - (4) Anal fissures, hematomas, or tears; perineal bruising
 - (5) Immediate reflex anal dilatation (>20 mm) in the absence of feces in the ampulla
 - c. The absence of physical findings does not exclude abuse; they are present in only 10% of cases of documented sexual abuse.
3. Laboratory evaluation
 - a. Cultures (not antigen assays) of:
 - (1) Vagina or urethra → for *Neisseria gonorrhoeae* or *Chlamydia trachomatis*
 - (2) Rectum → for *N gonorrhoeae*
 - (3) Throat → for *N gonorrhoeae*
 - b. Serologic testing for syphilis
 - c. HIV testing (after counseling) if infection is suspected
 - d. Pregnancy test if appropriate
4. Management
 - a. Gonococcal prophylaxis: indicated if abuse occurred <48 hours before evaluation

- b. Pregnancy prevention should be offered if there is a risk of pregnancy (ie, pubertal female with vaginal penetration).
- c. Cases of suspected sexual child abuse must be reported to child protective services or the police.

D. Physical abuse

1. 66% of cases occur in children <3 years old; 33% are <6 months old.
2. Most fatalities involve head injuries or abdominal trauma.
3. **"Red flags"**
 - a. History of injury is inconsistent with injury sustained.
 - b. History of injury is inconsistent with child's motor development.
 - c. Seeking of medical attention is delayed.
 - d. Explanation of the event keeps changing.
 - e. Presence of untreated injuries
 - f. Bruise marks over multiple areas, especially if over areas that are not typically injured accidentally (ear pinnae, low back, buttocks, thigh, ankles, wrists, neck, around mouth)
 - g. Certain types of patterned injuries (choke marks, gag marks, belt marks, tie marks, frenulum injuries, loop-shaped marks [electric cord or wire], linear marks)
4. Types of trauma associated with nonaccidental trauma
 - a. Any fractures in children <6 months old
 - b. Spiral fractures (from twisting an extremity), particularly when present in nonambulatory children
 - c. Metaphyseal chip or "corner" fractures
 - d. "Bucket handle" fractures
 - e. Rib fractures
 - f. Untreated healing fractures
 - g. Shaken baby syndrome
 - (1) Usually presents as unexplained seizures, abnormal behavior, unresponsiveness, or respiratory distress
 - (2) Triad of severe intracranial bleeding (subdural, intraparenchymal, interhemispheric, epidural), retinal hematomas (present in 70%–90% of cases), and no or minimal external signs of trauma
 - (3) Symptoms are the result of increased intracranial pressure.
 - (4) Associated posterior rib fractures are common.
 - h. Complex skull fractures
 - i. Duodenal hematomas from abdominal trauma
 - j. Cigarette burns
 - k. Immersion burns in a "glove-and-stockings" pattern
5. Diagnostic evaluation
 - a. Laboratory evaluation is guided by findings on examination and includes CBC, platelet count, and prothrombin time (INR)/partial thromboplastin time when the examination reveals multiple bruises and when there is any suggestion by the caregiver of easy bruisability.

b. Radiographic evaluation

- (1) A skeletal survey when evidence of trauma is noted
- (2) CT of the head when retinal hemorrhages or an acute deterioration in neurologic status is present
- (3) Abdominal/pelvic CT for abdominal wall bruising, unexplained vomiting or abdominal pain, shock, anemia, or abnormal liver function tests

6. Management

- a. Complete history and physical examination with verbatim quotations if possible
- b. Suspected cases must be reported to the police and/or child protection services.
- c. Hospitalization or placement in protective custody may be necessary to protect the child from further harm. Other children in the household must also be evaluated.
- d. Failure to report a suspected case may lead to misdemeanor charges and further harm or death to the child who is returned to the abusive environment.

PEDIATRIC EMERGENCIES: PRACTICE CLINICAL SCENARIOS

Answers immediately follow the practice clinical scenarios.

Scenario A

Presentation: A 4-year-old unimmunized child is sitting upright with chin thrust forward, mouth open, and neck slightly extended. He appears toxic and apprehensive and, on closer inspection, is drooling, with difficulty swallowing and stridor. The mother relates that the child was well until just a few hours ago, when he started to complain of a sore throat and developed a fever.

What is the diagnosis?

Scenario B

Presentation: A 2-year-old child has a barking, "seal-like" cough that the mother says is usually worse at night and sometimes causes severe respiratory distress. The child had a recent upper respiratory infection, and the cough, hoarseness, and stridor appeared just a few days after. The child does not have a fever and is nontoxic in appearance.

What is the diagnosis?

Scenario C

Presentation: A 2-year-old child has a high fever, stridor, and appears toxic. His mother reports that he has had nasal congestion, barking cough, and stridor for the past few days and then suddenly took a turn for the worse over the past few hours.

What is the diagnosis?

Scenario D

Presentation: A young child has had a mild fever, rhinorrhea, and conjunctivitis for the last week or two. She is now having numerous episodes of unremitting paroxysmal coughing followed by a "whoop." Vomiting often occurs in association with these paroxysms.

What is the diagnosis?

ANSWERS TO PRACTICE CLINICAL SCENARIOS

Scenario A

Diagnosis: epiglottitis

Diagnostic evaluation: If there is only slight respiratory distress, the diagnosis can be confirmed with a portable lateral soft-tissue radiograph of the neck. Findings include the "thumb print sign" and mild hypopharyngeal distention. Evaluation of the epiglottis by using a tongue blade (or a fiberoptic bronchoscope) should be avoided in children with any respiratory distress. Definitive diagnosis is ideally made in the operating room under controlled conditions by direct visualization of the "cherry-red" epiglottis. This is particularly true when severe respiratory distress is present.

Management: Disturb the child as little as possible to reduce anxiety, because he is at high risk of complete airway obstruction. Allow him to assume a position of comfort (usually sitting upright) and remain with his parents. Do *not* force him to lie supine.

Provide supplemental humidified oxygen, set up airway stabilization equipment at bedside, and obtain immediate ENT and anesthesia consults. Once the airway has been stabilized, draw blood for laboratory studies (including cultures), and administer parenteral antibiotics (cefuroxime, cefotaxime, ceftriaxone, or ampicillin-sulbactam). Admit to ICU.

Scenario B

Diagnosis: croup

Diagnostic evaluation: The diagnosis can often be made on clinical grounds, but radiographs can be useful to exclude epiglottitis. The PA neck radiograph may reveal symmetric subglottic narrowing of the tracheal air column ("steeple sign"), and the lateral neck radiograph may show a distended hypopharynx and subglottic narrowing. The epiglottis and retropharyngeal space are normal.

Management: Management is supportive, including cool mist, oxygen as needed, hydration (orally or IV), racemic epinephrine aerosol, steroids (one dose), and heliox if croup is severe or respiratory distress continues despite steroids and epinephrine. Consider admission if the child has persistent stridor at rest, cannot tolerate oral fluids, or has an incomplete response to epinephrine treatment.

Scenario C

Diagnosis: bacterial tracheitis

Management: Provide supplemental humidified oxygen, and obtain immediate consult with ENT and anesthesiology for endoscopy. Secure the airway, and obtain appropriate laboratory studies (including a Gram stain and culture of tracheal secretions). Administer IV hydration and antibiotics effective against *Staphylococcus aureus*. Admit to ICU.

Scenario D

Diagnosis: pertussis (whooping cough)

Diagnostic evaluation: A chest radiograph may show peribronchial thickening or a "shaggy" heart border. If the primary illness is complicated by pneumonia, infiltrates may be seen. The WBC count is $35,000/\text{mm}^3$ with a marked absolute lymphocytosis. The organism can be cultured using nasopharyngeal or sputum specimens, or detected with fluorescent antibody staining (or PCR) of a nasopharyngeal swab specimen.

Management: Begin erythromycin or azithromycin therapy and provide supplemental oxygen and postural drainage. Admit if the child has hypoxia and cyanosis during coughing spells or apnea. Monitor for pulmonary (eg, pneumonia, pneumothorax, hypoxia) and neurologic (eg, seizures, encephalitis) complications.

TOXICOLOGIC DISORDERS: SELF-ASSESSMENT QUESTIONS

1. All of the following are causes of a cholinergic toxidrome except:
 - (a) Organophosphates
 - (b) Sarin
 - (c) Cyclic antidepressants
 - (d) Carbamates
2. Methanol poisoning should be suspected when:
 - (a) The urine fluoresces strongly, and envelope-shaped crystals are seen on microscopy.
 - (b) The measured serum osmolality is 290 mOsm/L.
 - (c) Three teenagers present with visual complaints, and a fourth is found dead the day after a house party.
 - (d) An alcoholic patient presents with profound ketosis and a normal arterial blood gas after drinking from a bottle purchased at a hardware store.
3. GI hemorrhage is characteristic of which toxic ingestion?
 - (a) Lithium
 - (b) Iron
 - (c) Phosphorus
 - (d) Arsenic
4. The antidote for iron poisoning is:
 - (a) Dimercaprol
 - (b) Deferoxamine
 - (c) N-acetylcysteine
 - (d) Calcium disodium edetate (EDTA)
5. Severe salicylate poisoning (serum level ≥ 100 mg/dL) requires which of the following treatments immediately?
 - (a) N-acetylcysteine
 - (b) Acidification of the urine
 - (c) Charcoal hemoperfusion
 - (d) Hemodialysis
6. Clinical findings that indicate a serious overdose of a cyclic antidepressant include all of the following except:
 - (a) Ventricular dysrhythmias
 - (b) Wide QRS complex
 - (c) AV block
 - (d) Hypertension

7. All of the following would be appropriate for treatment of symptomatic organophosphate poisoning except:
- (a) Physostigmine
 - (b) Benzodiazepines
 - (c) Pralidoxime
 - (d) Atropine
8. A homeless man arrives at the emergency department appearing intoxicated. As part of his diagnostic evaluation, the following laboratory studies are ordered. What is this patient's serum osmolarity?
- | | | | |
|-----------------|------------|------------|-----------|
| Sodium | 125 mmol/L | BUN | 2.8 mg/dL |
| Potassium | 3.1 mmol/L | Creatinine | 1.8 mg/dL |
| Chloride | 100 mmol/L | Glucose | 360 mg/dL |
| CO ₂ | 22 mmol/L | Ethanol | 0.207 g/L |
- (a) 294
 - (b) 288
 - (c) 261
 - (d) 271
9. The treatment of choice for ventricular dysrhythmias secondary to phenothiazine overdose is:
- (a) Acidification of the urine
 - (b) Sodium bicarbonate
 - (c) Phenytoin
 - (d) Procainamide
10. Seizures commonly occur after overdose of which of the following?
- (a) Morphine
 - (b) Tramadol
 - (c) Oxycodone
 - (d) Fentanyl
11. Early signs and symptoms of ethanol withdrawal include:
- (a) Hypertension, tachycardia, and irritability
 - (b) Diaphoresis and dehydration
 - (c) Hypertension, tachycardia, and fever
 - (d) Visual hallucinations and paranoid ideation
12. Cocaine-induced cardiac dysrhythmias should be treated with any of the following medications except:
- (a) Benzodiazepines
 - (b) Sodium bicarbonate
 - (c) Calcium channel blockers
 - (d) β -blockers

13. An elderly patient who presents with unexplained CNS dysfunction and arterial blood gases that reveal a mixed acid-base disturbance should be suspected of having:
- (a) Acute salicylate poisoning
 - (b) Acetaminophen poisoning
 - (c) Chronic salicylate poisoning
 - (d) Iron poisoning
14. Indications for hemodialysis in patients with acute salicylate toxicity include all of the following except:
- (a) Severe acid-base imbalance
 - (b) Increasing serum ASA levels despite aggressive therapy
 - (c) Urine pH <6 and serum K^+ <3.0 despite IV sodium bicarbonate
 - (d) Serum ASA level >100 mg/dL in a patient without progressive signs and symptoms
15. In the clinical stages of iron poisoning, recovery from GI signs and symptoms classically occurs during:
- (a) Stage I
 - (b) Stage II
 - (c) Stage III
 - (d) Stage V
16. The aspirational hazard of a given hydrocarbon is greatest when it has two physical properties:
- (a) Low viscosity and low volatility
 - (b) High viscosity and low volatility
 - (c) Low viscosity and high volatility
 - (d) High viscosity and high volatility
17. With caustic ingestions, the degree of injury depends on all of the following except:
- (a) The type of ingestion (alkali or acid)
 - (b) The tone of the esophageal wall
 - (c) The volume and concentration of the ingested caustic
 - (d) The presence or absence of food in the stomach
18. Seizures are an indication for hemodialysis in all of the following poisonings except:
- (a) Digoxin
 - (b) Lithium
 - (c) Theophylline
 - (d) Methanol

19. Which of the following is potentially harmful if given to a fire victim with suspected cyanide poisoning?
- (a) Hydroxocobalamin
 - (b) Supplemental oxygen
 - (c) Amyl nitrate
 - (d) Sodium thiosulfate
20. Which of the following is false regarding button battery ingestions?
- (a) Immediate surgical consult should be obtained if GI tract perforation is suspected.
 - (b) If radiography demonstrates the battery is lodged in the esophagus, immediate endoscopic removal is indicated.
 - (c) If radiography demonstrates the battery has entered the stomach, the patient should be monitored on an outpatient basis until the battery has passed.
 - (d) If radiography demonstrates the battery has entered the stomach, immediate endoscopic removal is indicated.
21. Which is least appropriate for the management of asymptomatic pediatric sulfonylurea ingestions?
- (a) Frequent blood glucose testing (every 15–20 minutes)
 - (b) Empiric administration of glucose
 - (c) Thorough physical examination, looking for signs of hypoglycemia
 - (d) Hospital admission

ANSWERS

- | | | | | | |
|------|------|-------|-------|-------|-------|
| 1. c | 5. d | 9. b | 13. c | 17. b | 21. b |
| 2. c | 6. d | 10. b | 14. c | 18. a | |
| 3. b | 7. a | 11. a | 15. b | 19. c | |
| 4. b | 8. d | 12. d | 16. c | 20. d | |

Use the pre-chapter multiple choice question worksheet (page xx) to record and determine the percentage of correct answers for this chapter.

I. GENERAL APPROACH TO THE POISONED PATIENT

A. History

1. Determine the involved poison.
2. Determine route of exposure: ingestion, inhalation/insufflation, injection, dermal, etc.
3. Determine how much was taken and inquire about extended-release preparations.
4. Determine when it was taken.
5. Determine whether the exposure was accidental or intentional; evaluate for suicidality.
6. Determine what the patient was doing at the time he or she became ill. Examples:
 - a. Fumigating a ship → cyanide
 - b. Working in a garage → carbon monoxide, pesticides, toxic alcohols
 - c. Applying chemicals to crops → organophosphates/carbamates

B. Physical examination

Table 29: Physiologic Effects Associated with Specific Poisons

Poison	Vital Signs	Level of Consciousness
Cyclic antidepressants	↑ pulse ^a ↓ blood pressure ↓ respiration	↓
Barbiturates	↓ blood pressure ↓ respiration ↓ temperature ^b	↓ ^a
Phenothiazines (eg, chlorpromazine, prochlorperazine)	↑ pulse ↓ blood pressure ↓ temperature	↓
Digitalis	↓ pulse ^a ↓ blood pressure	
Opioids	↓ pulse ↓ blood pressure ^a ↓ respiration ↓ temperature	↓
Clonidine	↓ pulse ^a ↑ blood pressure early (uncommon), then ↓ blood pressure later ↓ respiration (uncommon) ↓ temperature	↓ (mostly in children)
Ethanol, ethylene glycol, methanol, isopropanol	↑ pulse ↓ blood pressure ↓ respiration ^{a, c} ↓ temperature ^b	↓

Poison	Vital Signs	Level of Consciousness
Cocaine	↑ pulse ↑ blood pressure ↑ or ↓ respiration ↑ temperature	
Amphetamines	↑ pulse ↑ blood pressure ↑ temperature	
Phencyclidine (PCP)	↑ pulse ↑ blood pressure ↑ temperature	
Salicylates	↑ respiration ↑ temperature ^a	↓
Iron	↓ blood pressure	
Liquid petroleum distillates (gasoline, kerosene)	↑ pulse ↑ respiration ^a ↑ temperature	
Anticholinergics	↑ pulse ^a ↑ temperature	↓
Organophosphates/ carbamates	↑ pulse early ^a , then ↓ pulse later ↑ or ↓ blood pressure ↑ respiration ↓ O ₂ saturation	↓
Sedative-hypnotics	↓ pulse ↓ blood pressure ↓ temperature ^b	↓ ^a

a Earliest sign

b Poisoning can cause cutaneous vasodilation, resulting in poikilothermia. Because the ambient temperature is generally lower than body temperature, the more common clinical manifestation is hypothermia. Be aware that poisoning while exposed to high ambient temperatures (eg, unconscious victim in a hot car) can manifest hyperthermia by the same mechanism.

c Depressed respiratory rate is an early sign associated with intoxication by various alcohols. In cases of ethylene glycol and methanol poisoning, when metabolic acidosis may occur, hyperventilation may occur as a compensatory mechanism and is expected to be a later finding.

C. Toxidromes

1. Cholinergic (wet manifestations)

a. Clinical presentation

(1) "DUMBBELS"

Diarrhea

Urination

Miosis

Bradycardia

Bronchospasm/bronchorrhea

Emesis

Lacrimation

Salivation

- (2) **Seizures, coma, and respiratory failure**
- (3) **Muscle fasciculations and weakness**
- b. **Examples**
 - (1) **Organophosphates**
 - (a) **Insecticides (eg, parathion, diazinon)**
 - (b) **Nerve agents (sarin, soman, VX)**
 - (2) **Carbamates**
 - (a) **Insecticides (eg, carbaryl)**
 - (b) **Physostigmine, pyridostigmine (mostly peripheral symptoms)**
 - (3) **Muscarine-containing mushrooms (*Clitocybe*, *Inocybe*)**
- 2. **Anticholinergic (dry manifestations)**
 - a. **Clinical presentation**
 - (1) **Mad as a hatter (delirium), hot as a hare (hyperthermia), blind as a bat (loss of ability to accommodate near vision and mydriasis), dry as a bone (dry mucous membranes, urinary retention, decreased bowel sounds), red as a beet (flushing), tacky as a leisure suit (tachycardia)**
 - (2) **Seizures**
 - b. **Examples**
 - (1) **Cyclic antidepressants**
 - (2) **Atropine, scopolamine**
 - (3) **Antihistamines**
 - (4) **Jimsonweed**
- 3. **Opioid drugs**
 - a. **Clinical presentation**
 - (1) **Clinical triad: coma, respiratory depression, and miosis**
 - (2) **Miosis does not always occur (eg, meperidine, propoxyphene, diphenoxylate and atropine).**
- 4. **Sedative-hypnotics**
 - a. **Clinical presentation: CNS and respiratory depression**
 - b. **Examples: barbiturates, ethanol, benzodiazepines, gamma hydroxybutyric acid, others**
- 5. **Sympathomimetics**
 - a. **Clinical presentation**
 - (1) **CNS stimulation**
 - (2) **May lead to toxic psychosis; presence of abnormal vital signs (increased blood pressure, wide pulse pressure, hyperthermia), disorientation, and clouding of consciousness help differentiate from psychiatric conditions (schizophrenia).**
 - b. **Examples: amphetamines, cocaine**
- 6. **Hallucinogens**
 - a. **Clinical presentation**
 - (1) **Hallucinations or cognitive disorders, although patients remain oriented to person, place, and time**
 - (2) **Tachycardia, hypertension, and mydriasis**

7. Extrapyrarnidal
 - a. Clinical presentation: a "parkinsonian" picture: **TROD**
 - Tremor, torticollis, trismus
 - Rigidity
 - Opisthotonos, oculogyric crisis
 - Dysphonia, dysphagia
 - b. Examples: all of the "-zines" (chlorpromazine, promethazine, etc), haloperidol, metoclopramide
8. Serotonin syndrome (mimics neuroleptic malignant syndrome)
 - a. Clinical presentation
 - (1) Altered mental status, seizures, coma
 - (2) Muscle rigidity (especially myoclonus)
 - (3) Autonomic instability (hyperthermia, hypertension, tachycardia, tachypnea)
 - b. Usually precipitated by overdose or drug-drug interaction between several serotonergic agents: SSRIs, monoamine-oxidase inhibitors, lithium, meperidine, or dextromethorphan
9. Hemoglobinopathies
 - a. Carboxyhemoglobinemia—clinical presentation
 - (1) Headache
 - (2) Nausea, vomiting, flu-like syndrome
 - (3) Syncope, tachypnea, tachycardia, chest pain
 - (4) Confusion, coma, convulsions
 - (5) Cardiovascular collapse, respiratory failure
 - b. Methemoglobinemia—clinical presentation
 - (1) Cyanotic appearance (often more severe than would be expected based on degree of methemoglobinemia)
 - (2) Chocolate-colored blood despite exposure to air
 - (3) Asymptomatic (healthy patients with <20% methemoglobin level)
 - (4) Fatigue, weakness, dizziness, headache, tachycardia (20%–40%)
 - (5) Lethargy, stupor, respiratory depression, acidosis (40%–50%)
10. Metal fume fever
 - a. Acute illness that occurs when the fumes of certain metals are inhaled (primarily zinc, copper, magnesium, cadmium, nickel, brass, iron, manganese, and tin)
 - b. An occupational hazard to welders and workers in foundries, marinas, and shipyards
 - c. Clinical presentation
 - (1) Fever, shaking chills, excessive salivation, metallic taste in mouth, headache, cough, and respiratory distress
 - (2) Signs and symptoms may be delayed 4–8 hours after exposure and are usually resolved within 24–48 hours.
 - d. Management: supportive, including bronchodilators if wheezing
11. Withdrawal syndromes that may be confused with toxidromes
 - a. Ethanol or sedative-hypnotic withdrawal
 - (1) Clinical presentation: tremor, visual hallucinations, agitation, confusion, disorientation, autonomic overactivity, and seizures

- (2) Immediate pharmacologic goal is adequate sedation with another sedative (usually benzodiazepines).
- (3) Other therapy: hydration, glucose, thiamine; magnesium and phosphorous replacement may also be necessary.
- b. Opioid withdrawal
 - (1) Clinical presentation: restlessness, mydriasis, lacrimation, gooseflesh, yawning, insomnia, diarrhea, abdominal pain, and muscle cramps
 - (2) Patient is appropriately oriented, unlike in withdrawal from sedative-hypnotics, and seizures do not occur (except in neonates).
 - (3) Management: supportive (antiemetics, nonopioid analgesics for muscle cramps and abdominal pain)

D. Major toxic signs

1. Cardiac dysrhythmia (always consider obtaining an ECG in poisoned patients)
 - a. Prolonged QT interval → phenothiazines, lithium, class IA (quinidine, procainamide), class IC (encainide, flecainide, propafenone), and class III (amiodarone, sotalol) antidysrhythmics, antidepressants, and methadone
 - b. Wide QRS complex → cyclic antidepressants, quinine, quinidine, class IA and IC drugs, propoxyphene, cocaine, carbamazepine, amantadine, antihistamines, chloroquine, phenothiazines
 - c. Sinus bradycardia → digitalis, cyanide, organophosphates, carbamates, β -blockers, calcium channel blockers, clonidine, opioids, sedative-hypnotics
2. Metabolic acidosis
 - a. Calculate the anion gap (the difference between the positive and negative organic ions).
 - (1) $\text{Na}^+ - (\text{Cl}^- + \text{HCO}_3^-)$
 - (2) Normal value = 12 ± 4 mmol/L
 - (3) Differential diagnosis of anion gap metabolic acidosis:
 - Methanol, metformin
 - Uremia
 - Diabetic or alcoholic ketoacidosis
 - Paraldehyde, phenformin
 - Isoniazid, iron, or inhalants (carbon monoxide, cyanide, hydrogen sulfide)
 - Lactic acid
 - Ethylene glycol (ethanol may produce a small gap)
 - Salicylates, solvents
 - (4) Poisonings with a low anion gap (<8 mEq/L): lithium, bromides
 - b. Calculate the osmolar gap (the difference between the measured serum osmolality and the calculated osmolality).
 - (1) Normal osmolar gap: ± 10
Normal serum osmolality: 280–295 mOsm
 - (2) Formula for calculating osmolality

$$(2 \times \text{Na}) + \frac{\text{BUN}}{2.8} + \frac{\text{Glucose}}{18} + \frac{\text{Ethanol}}{4.6}$$

(3) Osmolar gap ≥ 10 is consistent with poisoning by alcohols (ethanol, methanol, ethylene glycol, isopropanol, propylene glycol), glycerol, or mannitol.

(4) Absence of an osmolar gap does not exclude toxic alcohol poisoning!

3. Coma—differential diagnosis of pinpoint pupils
 - a. Opioids
 - b. Phenothiazines
 - c. Clonidine
 - d. Sedative hypnotics, barbiturates
 - e. Organophosphates, carbamates, parasympathomimetics
 - f. Phencyclidine (PCP)
 - g. Nicotine
 - h. Cholinergic agents
 - i. Brainstem lesions (pontine hemorrhage)
4. Hyperthermia
 - a. Serotonin syndrome
 - b. Neuroleptic malignant syndrome
 - c. Salicylates
 - d. Sympathomimetics
 - e. Malignant hyperthermia
5. GI symptoms
 - a. Iron \rightarrow severe vomiting, GI bleeding, diarrhea
 - b. Lithium \rightarrow nausea, vomiting, diarrhea
 - c. Mercury \rightarrow salivation, diarrhea, GI hemorrhage
 - d. Phosphorus \rightarrow luminescent vomitus, flatus, smoking feces
 - e. Arsenic, colchicine, fluoride \rightarrow vomiting, diarrhea, oropharyngeal pain
 - f. Mushrooms \rightarrow nausea, vomiting, diarrhea
 - g. Opioid withdrawal, laxative abuse \rightarrow diarrhea, abdominal pain
 - h. Organophosphates, carbamates, nicotine \rightarrow nausea, vomiting, diarrhea
 - i. Theophylline \rightarrow severe vomiting
 - j. Acetaminophen, carbon tetrachloride, amatoxin \rightarrow hepatitis
 - k. Black widow spider \rightarrow abdominal wall rigidity
 - l. Boric acid, copper \rightarrow blue-green emesis
 - m. Digitalis, ASA, NSAIDs, carbon monoxide \rightarrow nausea, vomiting
6. Seizures
 - a. Anticholinergics (cyclic antidepressants, antihistamines)
 - b. Phencyclidine (PCP)
 - c. LSD
 - d. Stimulants (cocaine, amphetamines, theophylline)
 - e. Sedative-hypnotic withdrawal
 - f. Carbon monoxide
 - g. Opioids (meperidine, tramadol)
 - h. Disulfiram

- i. Organophosphates
- j. Camphor
- k. Phenothiazines
- l. Isoniazid (classic board scenario is status epilepticus)
- m. Monomethylhydrazine-containing mushrooms (*Gyromitra*) (classic board scenario is status epilepticus)
- n. Carbamazepine
- o. Monoamine-oxidase inhibitors (toxicity or drug/food interactions)
- p. Lindane
- q. Nicotine
- r. Gamma hydroxybutyrate
- 7. Seizure mimics: strychnine, tetanus
- 8. Pulmonary edema
 - a. Opioids
 - b. Salicylates
 - c. Sedative-hypnotics
 - d. Carbon monoxide
 - e. Cardiac drugs: digoxin, β -blockers, calcium channel blockers
 - f. Toxic inhalations: chlorine, N_2O , phosgene, zinc chloride
 - g. Cocaine
 - h. Organophosphates
- 9. Breath odor
 - a. Isopropyl alcohol \rightarrow fruit-like
 - b. Phenols, creosol \rightarrow disinfectants
 - c. Cyanide \rightarrow bitter almonds or silver polish
 - d. Chloral hydrate \rightarrow pear
 - e. Organophosphates, arsine, phosphorus \rightarrow garlic
 - f. Nitrobenzene \rightarrow shoe polish, aniline dye
 - g. Turpentine \rightarrow violets
 - h. Carbon tetrachloride \rightarrow cleaning fluid
 - i. Ethchlorvynol \rightarrow new vinyl shower curtain
 - j. Hydrogen sulfide \rightarrow rotten eggs
 - k. Camphor, naphthalene, p-dichlorobenzene \rightarrow mothballs
 - l. Phosgene \rightarrow hay
 - m. Methyl salicylate \rightarrow wintergreen
- 10. Skin findings
 - a. Needle tracks: IV drug use
 - b. Pressure sores and bullae: barbiturates, carbon monoxide, sedative-hypnotics (snake and spider bites also cause bullae)
 - c. "Boiled lobster" skin: boric acid (roach tablets)
 - d. Diaphoresis: salicylates, organophosphates, sympathomimetics, cholinergics, ethanol or sedative-hypnotic withdrawal

- e. Jaundice: acetaminophen, mushroom poisoning, arsine gas
- f. Alopecia: arsenic, thallium, cytotoxic agents
- g. Flushing: disulfiram, niacin, ethanol, monoamine-oxidase inhibitors, metronidazole, anticholinergics, scombroid fish poisoning
- h. Cyanosis (noncardiopulmonary): nitrates/nitrites, "-caine" anesthetics, aniline dyes, chlorates, dapsone, sulfonamides

11. Alterations in motor tone

- a. Increased motor tone: lithium, levodopa, strychnine, phencyclidine (PCP), extrapyramidal, black widow spider bite, serotonin syndrome
- b. Fasciculations: cholinergics, amphetamines, heavy metals, paralytic shellfish poisoning, scorpion, or pufferfish
- c. Flaccid: sedative-hypnotics, opioids, fasciculants listed above

12. Bowel sounds

- a. Increased bowel sounds: cathartics (eg, sorbitol), cholinergics, heavy metals, mushrooms, withdrawal syndromes
- b. Decreased bowel sounds: opioids, anticholinergics, phenothiazines

E. Diagnostic evaluation

1. ECG → dysrhythmias and conduction blocks
2. CBC → as indicated for specific poisons (eg, iron)
3. Electrolytes → for calculating osmolar and anion gaps
4. Glucose → hyper/hypoglycemia and osmolar gap determinations
5. Serum osmolality → osmolar gap determination
6. BUN/creatinine → osmolar gap determination (BUN), renal function
7. Creatine kinase → as clinically indicated to evaluate for rhabdomyolysis
8. Arterial blood gases → acid-base evaluation
9. Specific drug levels: salicylate, acetaminophen, and ethanol, plus any other toxic agent that is suggested by the history and physical examination (findings uncovered in the the history and physical examination frequently point to a specific toxic agent/class)
10. Abdominal radiographs → look for radiopaque tablets ("CHIPES"); some poisons that can be radiopaque:

Chloral hydrate

Heavy metals

Iron

Phenothiazines

Enteric-coated tablets

Solvents

11. Urinalysis

- a. Urine pH as indicated for management of specific poisonings (eg, salicylates)
- b. Ketones → isopropanol, alcoholic/starvation ketoacidosis, salicylates
- c. Blood without RBCs → rhabdomyolysis (eg, cocaine)
- d. Crystal fluorescence → ethylene glycol (not reliable in practice)

F. General treatment measures

1. GI decontamination

a. Activated charcoal

- (1) Action: binds the toxic agent and prevents absorption
- (2) Dose: 1 g/kg or 10 g per gram of ingested toxin, whichever is greater
- (3) Not useful for the following ingestions:
 - (a) Lithium
 - (b) Alkalis/acids (absolutely contraindicated)
 - (c) Heavy metals
 - (d) Iron
 - (e) Alcohols
 - (f) Hydrocarbons and solvents
- (4) Repeated doses of activated charcoal (0.5 g/kg every 4–6 hours) are used for poisoning due to salicylates, theophylline, barbiturates, valproic acid, carbamazepine, dapsone, and sustained-release preparations.
 - (a) Should not be given to patients suspected of having a bowel obstruction or ileus (confirm presence of bowel sounds before administration)
 - (b) Should not be accompanied by repeated doses of a cathartic (eg, sorbitol), which can cause severe fluid and electrolyte disturbances

b. Whole-bowel irrigation (WBI)

- (1) Uses polyethylene glycol electrolyte lavage solution (PEG-ELS) to wash poisons from GI tract before they are absorbed
- (2) May be useful after ingestions of:
 - (a) Lithium
 - (b) Iron
 - (c) Heavy metals
 - (d) Sustained-release, highly toxic drugs (eg, verapamil, diltiazem, theophylline, bupropion)
 - (e) Body packers/stuffers
- (3) Must administer via nasogastric tube (volume required is too large for patient to drink independently): adults = 1,500–2,000 mL/hr; children = 25–40 mL/kg/hr
- (4) Activated charcoal can be used before and during WBI therapy.
- (5) End point is retrieval of all pills or packets from body packers/stuffers. Other commonly cited end points include presence of clear rectal effluent; however, WBI should continue despite clear rectal effluent if pills or packets are still present in GI tract.
- (6) Contraindications
 - (a) Unprotected airway
 - (b) Intestinal obstruction or ileus
 - (c) Uncertainty regarding position of nasogastric tube
 - (d) Intractable vomiting

- c. GI decontamination measures no longer recommended
 - (1) Neutralization of acids or bases: produces exothermic reaction that can cause further tissue damage
 - (2) Cathartics (to promote rapid transit of toxin through the GI tract): no proven benefit and can cause fluid and electrolyte imbalances
- 2. Removal of unabsorbed poison assumes a secondary role and, in many cases, may not be performed.
 - a. Induction of emesis
 - (1) Not indicated for prehospital or in-hospital therapy
 - (2) American Academy of Pediatrics no longer recommends ipecac be kept in the home.
 - b. Gastric lavage
 - (1) Indications
 - (a) Known or suspected life-threatening ingestion and
 - (b) Time of ingestion <1 hour before arrival in emergency department or drug still present in stomach
 - (2) Procedure
 - (a) Patients should be intubated before lavage in nearly all cases.
 - (b) Place patient on left side and in Trendelenburg to minimize flushing of the poison into the small intestine.
 - (c) Use a #36–40 French Ewald orogastric tube for adults, #15–28 French orogastric tube for children. (Use of smaller tubes may not recover larger pill fragments).
 - (d) Lavage with aliquots of 200 mL of warm tap water or isotonic saline until clear (adults); use aliquots of 10 mL/kg in children.
 - (3) Contraindications
 - (a) Unprotected airway
 - (b) Ingestion of caustic agents or low-viscosity hydrocarbons
 - (c) Nontoxic ingestion
- 3. Elimination of absorbed toxin
 - a. Antidotes

Table 30: Antidotes for Specific Poisons

Toxin	Antidote(s)
Acetaminophen	N-acetylcysteine
Arsenic	British antiLewisite (BAL; dimercaprol) or 2,3-dimercaptosuccinic acid (DMSA)
Mercury	
Lead	
Carbamates	Atropine
Carbon monoxide	Oxygen Hyperbaric oxygen
Cyanide	Amyl nitrite pearls Sodium nitrite (3% solution) Sodium thiosulfate (25% solution) Hydroxycobalamin
Ethylene glycol	Ethanol (10%) mixed in D5W
Methanol	Fomepizole (4-methylpyrazole)
Iron	Deferoxamine
Lead	Calcium disodium edetate (EDTA)
Methemoglobin-forming agents	Methylene blue (1% solution)
Organophosphate	Atropine Pralidoxime (2-PAM)
Opioids	Naloxone
Phenothiazines	Diphenhydramine Benztropine
Isoniazid (INH)	Pyridoxine Benzodiazepines as needed for seizures
Digoxin	Digoxin-specific Fab fragments
Digitoxin	
Oleander	
Benzodiazepines	Flumazenil (use is controversial: seizures can occur; contraindicated if QRS >100 milliseconds, benzodiazepine-dependent patient, or coingested cyclic antidepressant)

b. Acidification of the urine is no longer recommended.

c. Alkalinization therapy with IV sodium bicarbonate

- (1) Cyclic antidepressants and other sodium-channel blockers → alkalinization of the blood helps improve/prevent dysrhythmias/conduction blocks and improves hypotension

- (2) Alkalinization of the urine to a pH >7 ionizes weak acids into ionized molecules, increasing excretion of the following toxic agents:
 - (a) Salicylates
 - (b) Phenobarbital
 - (c) Chlorpropamide
- d. Charcoal hemoperfusion rarely used any longer
- e. Hemodialysis indications
 - (1) Ethylene glycol
 - (2) Methanol
 - (3) Valproic acid
 - (4) Lithium
 - (5) Salicylates
 - (6) Theophylline, caffeine

G. Latent complications

1. Hepatotoxicity → acetaminophen, iron, carbon tetrachloride, cyclopeptide mushrooms (*Amanita*, *Galerina*, *Lepiota*), and pennyroyal oil
2. GI hemorrhage
 - a. ASA
 - b. Iron
 - c. Coumadin
 - d. Mercuric salts
 - e. Alkalis/acid caustics
 - f. Long-acting anticoagulant rodenticides
3. Renal failure → carbon tetrachloride, mercuric salts, acetaminophen, colchicine, ethylene glycol
4. Dysrhythmias → cyclic antidepressants, cocaine, β -blockers, calcium channel blockers, quinine, antidysrhythmics, chloroquine, propoxyphene, phenothiazine, theophylline, digoxin, chloral hydrate
5. Hypertension → phencyclidine (PCP), monoamine-oxidase inhibitors, phenylpropanolamine, cocaine, amphetamines
6. Hypotension → sustained-release calcium channel blockers
7. Neurotoxicity → botulinum toxin, lithium, carbon monoxide, methanol, carbon disulfide
8. Respiratory problems → nitrogen oxides, phosgene, paraquat

II. SPECIFIC OVERDOSES AND POISONINGS

A. Cyclic antidepressants

1. Commonly prescribed
 - a. Imipramine
 - b. Desipramine
 - c. Amitriptyline
 - d. Nortriptyline
 - e. Doxepin

2. Pathophysiology (obtain cardiac monitoring and ECG early)
 - a. Cardiac effects
 - (1) Anticholinergic activity that can induce tachycardia
 - (2) Quinidine-like activity (sodium and potassium channel blockade) that can induce intraventricular and AV blocks; avoid use of procainamide, quinidine, or other class IA and IC antiarrhythmics.
 - (a) Bundle-branch and fascicular blocks are usually preceded by a widening QRS complex.
 - (b) AV blocks range from first degree to complete block.
 - (3) Hypotension due to peripheral α -adrenergic blockade, dysrhythmias, and norepinephrine depletion
 - (4) Pulmonary edema
 - b. CNS effects
 - (1) Confusion, agitation, and hallucinations with rapid progression to coma
 - (2) Seizures are common and usually single.
 - (3) Physical examination findings may include:
 - (a) Clonus
 - (b) Choreoathetosis
 - (c) Myoclonic jerks
 - c. Anticholinergic effects may or may not occur; their absence does not exclude toxicity.
 - d. Signs that indicate a serious overdose
 - (1) Ventricular dysrhythmias
 - (2) Intraventricular conduction defects with a QRS complex >100 milliseconds
 - (3) Bradycardia and AV blocks
 - (4) Hypotension
 - (5) Pulmonary edema
 - (6) Seizures
 - (7) Cardiac arrest
3. Management
 - a. Activated charcoal: use with caution because patients have the potential to rapidly decompensate.
 - b. Consider orogastric lavage.
 - c. IV bolus of sodium bicarbonate is the mainstay of therapy.
 - (1) The most effective therapy for improving hypotension and abolishing dysrhythmias
 - (2) Indications: if QRS complex ≥ 100 milliseconds in the limb leads
 - (3) Administration of sodium bicarbonate is usually via boluses: dosing 1–2 mEq/kg (1–2 ampules of NaHCO_3).
 - (4) Avoid if arterial pH >7.55 or serum sodium >150 mmol/L.
 - d. Hypotension
 - (1) Start with crystalloids and sodium bicarbonate.
 - (2) If no or poor response, try drug therapy: norepinephrine or dopamine may be effective early in toxicity.

- e. Seizure control is important, because lactic acidosis can worsen cardiac toxicity. Therapeutic choices include:
 - (1) Sodium bicarbonate
 - (2) Benzodiazepines
 - (3) Phenobarbital
 - (4) Propofol
- f. Therapies to avoid
 - (1) Class IA (eg, procainamide) and IC (eg, flecainide) antidysrhythmics → can worsen the "quinidine-like" toxicity on the myocardium
 - (2) Flumazenil → risk of precipitating seizures
 - (3) Physostigmine → risk of cardiac toxicity and asystole
 - (4) Hemodialysis → ineffective because cyclic antidepressants are highly protein bound
 - (5) Hemoperfusion

B. Other antidepressants

1. Differ from the cyclic antidepressants in that their mechanism of action (inhibition of biogenic amine reuptake in the CNS) occurs without sodium channel blockade; therefore, life-threatening toxicity is less common.
2. Common agents
 - a. SSRIs: selective inhibition of serotonin reuptake
 - (1) Fluoxetine
 - (2) Sertraline
 - (3) Paroxetine
 - (4) Fluvoxamine
 - (5) Citalopram
 - b. Venlafaxine: serotonin-norepinephrine reuptake inhibitor
 - c. Trazodone, nefazodone: inhibit 5-hydroxytryptamine (5-HT) reuptake and block postsynaptic 5-HT₂ receptors; trazodone has peripheral alpha effects that can result in priapism when taken therapeutically or in overdose.
 - d. Bupropion: inhibits dopamine reuptake
 - e. Mirtazapine: nonselective reuptake of biogenic amines inhibited and blocks postsynaptic 5-HT₂ and 5-HT₃ receptors
3. Clinical presentation
 - a. SSRI overdose
 - (1) Mild CNS depression, sinus tachycardia, GI symptoms (nausea, vomiting, diarrhea), and seizures (rare)
 - (2) Serotonin syndrome
 - b. Bupropion overdose: hallucinations, tachycardia, and seizures (which may be delayed up to 18 hours after overdose)
 - c. Trazodone, nefazodone: prolonged QT interval
4. Management
 - a. Activated charcoal
 - b. Monitor for 6 hours—longer for bupropion and extended-release preparations
 - c. Benzodiazepines for seizures (rare)

d. Serotonin syndrome

- (1) Critical care monitoring
- (2) IV crystalloid therapy
- (3) Active cooling therapy for hyperthermia
- (4) Cyproheptadine 4–8 mg orally every 8–12 hours
- (5) Muscle rigidity must be controlled quickly, because it contributes to the hyperthermia, rhabdomyolysis, and lactic acidosis.
- (6) Evaluate for underlying medical complications (including infections).
- (7) Watch for hyperkalemia and myoglobinuric-induced renal failure secondary to rhabdomyolysis.

C. Lithium

1. Primary therapeutic use is for bipolar disorder.
2. Toxicity can develop after acute overdose or in a patient taking usual therapeutic dose who becomes dehydrated or develops renal insufficiency (chronic).
3. Clinical presentation
 - a. Toxicity is characterized primarily by neurologic manifestations
 - (1) Mild: fatigue, tremor, mild confusion
 - (2) Moderate: increased tremor, slurred speech, ataxia, increased confusion, drowsiness, hyperreflexia
 - (3) Severe: tremor, clonus, choreoathetosis, rigidity, hyperthermia, irritability, stupor, coma, seizures
 - (4) The normal therapeutic blood level of lithium is 0.6–1.2 mEq/L. Signs and symptoms of toxicity often do not correlate with serum levels (especially with chronic toxicity, because the lithium has shifted into the CNS [tissue] compartment); the neurologic status of the patient is the most important determinant of toxicity.
 - b. Other
 - (1) Nausea, vomiting, diarrhea
 - (2) Nephrogenic diabetes insipidus (chronic)
4. Diagnostic evaluation
 - a. Lithium level
 - b. ECG
 - (1) ST–T wave abnormalities
 - (2) Prolonged QT interval
 - (3) Dysrhythmias (rare)
5. Management
 - a. General measures
 - (1) Cardiac monitor
 - (2) Whole-bowel irrigation (acute overdoses, especially with sustained-release preparations)
 - b. Specific therapy
 - (1) The key to the treatment of lithium overdose is awareness of its mechanism of excretion.

- (a) 97% is excreted unchanged in the urine.
- (b) 75%–80% is reabsorbed by the proximal tubule along with sodium; volume depletion enhances lithium retention.
- (2) Restoration of sodium and water balance with normal saline.
 - (a) The choice of fluid and rate of administration should be based on the degree of dehydration and hypotension as well as the serum sodium level.
 - (b) Normal saline should be used unless the patient is severely hypernatremic
 - (c) Goal: correct dehydration and maintain urinary output at 1–2 mL/kg/hr.
- (3) Because lithium is reabsorbed in the proximal renal tubule, diuretics that act on the ascending limb of the loop of Henle (furosemide) or distal tubule (thiazides) will not increase lithium excretion and may promote reabsorption if they lead to volume depletion.
- (4) Indications for hemodialysis
 - (a) Clinical signs of moderate to severe poisoning (eg, seizure)
 - (b) Worsening clinical condition despite falling lithium levels
 - (c) Onset of dysrhythmias
 - (d) Decreasing urine output or renal failure (increased BUN/creatinine)
 - (e) Serum level ≥ 4.0 mEq/L at least 6 hours after an acute ingestion (controversial)
- (5) Watch for a rebound in serum lithium levels after hemodialysis, because tissue levels reequilibrate with the serum. Repeat hemodialysis is often necessary in chronic toxicity.
- (6) Failure to recognize or provide appropriate treatment may result in permanent neurologic sequelae.

D. Sedative-hypnotics

1. Barbiturates

a. Classification

- (1) Ultrashort acting
 - (a) Methohexital
 - (b) Thiopental
- (2) Short acting
 - (a) Pentobarbital
 - (b) Secobarbital
- (3) Intermediate acting
 - (a) Amobarbital
 - (b) Butabarbital
- (4) Long acting
 - (a) Phenobarbital
 - (b) Mephobarbital
 - (c) Primidone

b. Metabolism and major excretion mechanisms

- (1) Ultrashort-, short-, and intermediate-acting barbiturates are lipid-soluble and poorly ionized.
- (2) Long-acting barbiturates are not lipid-soluble, but they are ionized at an alkaline pH.

- (a) When ionized, drugs cannot enter neuronal cells or the resorptive cells that line the renal tubules.
 - (b) This is why alkalinization of the urine is therapeutic in phenobarbital overdose.
 - c. Clinical presentation
 - (1) Coma
 - (2) Hypothermia
 - (3) Respiratory depression
 - (4) Shock
 - (5) Pulmonary edema
 - (6) Absent corneal and deep tendon reflexes
 - (7) Cutaneous bullae
 - d. Potential for fatality is low in the absence of coingested CNS depressants; however, beware that they are synergistic with other CNS depressants.
 - e. Management
 - (1) General measures
 - (a) Airway management
 - (b) Fluid challenge if hypotension develops
 - (c) Activated charcoal
 - (2) Specific therapy of phenobarbital overdose
 - (a) Alkalinization of the urine with sodium bicarbonate can increase the excretion of phenobarbital 5- to 10-fold.
 - i. Goal: urine pH 7.5–8
 - ii. Use with caution, if at all, in patients dependent on phenobarbital (eg, those with alcohol dependence or seizure disorders). In these patients, supportive measures may be best.
 - (b) One method of urinary alkalinization
 - i. Begin therapy with a bolus of 1–2 mEq/kg (1–2 standard ampules; optional)
 - ii. Follow with a continuous infusion of 150 Eq NaHCO_3 (3 standard ampules) in 1,000 mL D5W and infuse at 1.5–2 times the maintenance fluid rate.
 - iii. Adjust infusion rate as indicated to achieve goal urine pH.
2. Benzodiazepines
- a. Agents within this class include diazepam, lorazepam, midazolam, alprazolam, chlordiazepoxide, clonazepam, flurazepam, flunitrazepam ("roofies"), oxazepam, temazepam, and triazolam.
 - b. Pathophysiology
 - (1) Potency, duration of effects, and active metabolites vary widely.
 - (2) Potential for fatality is low in the absence of coingested substances, but they are synergistic with other CNS depressants.
 - (3) Benzodiazepines enhance effects of gamma-aminobutyric acid (GABA) at central receptors.
 - c. Clinical presentation
 - (1) Progressive depression of level of consciousness to coma
 - (2) Hypothermia, pulmonary aspiration, and respiratory arrest

- d. Management
 - (1) General supportive measures: careful monitoring with attention to airway potency, oxygenation, and ventilatory support
 - (2) Flumazenil: a competitive antagonist for benzodiazepines at the receptor level
 - (a) Generally reserved for iatrogenic overdoses in non-benzodiazepine-dependent patients or pediatric exposures requiring active airway intervention (uncommon)
 - (b) Contraindicated in benzodiazepine-dependent patients, because it can induce withdrawal (may be severe and life-threatening)
 - (c) Contraindicated when coingestion with cyclic antidepressants is a possibility, because seizures may occur.
 - (d) Re-sedation is common (short half-life of the drug)
3. Gamma hydroxybutyrate
 - a. Epidemiology
 - (1) Youth rave culture
 - (2) Drug-facilitated sexual assault
 - (3) Bodybuilders
 - b. Clinical presentation
 - (1) Overdose
 - (a) Bradycardia, respiratory depression
 - (b) Lethargy, coma
 - (c) Miosis, myoclonus, nystagmus, seizures
 - (2) Withdrawal may resemble ethanol or sedative-hypnotic withdrawal (including delirium).
 - c. Management
 - (1) Overdose: supportive care, airway protection, intubation based on level of consciousness; *typical course is abrupt awakening within a few hours.*
 - (2) Classic scenario: Comatose patient presents and is intubated; hours later awakes abruptly, pulls out endotracheal tube and flees the emergency department.
 - (3) Withdrawal: benzodiazepines; pentobarbital for refractory cases
- E. Phenothiazines (eg, chlorpromazine, prochlorperazine)
 1. Pathophysiology
 - a. Erratic and unpredictable absorption after ingestion
 - b. Half-life is 20–40 hours, and effects may persist for days.
 - c. Metabolized in the liver
 2. Adverse effects seen with therapeutic doses and drug abuse
 - a. Extrapyramidal reactions (most common)
 - (1) Parkinsonian (rigidity, tremor)
 - (2) Akathisia (incessant movement and restlessness)
 - (3) Dystonic (grimacing, trismus, torticollis, dysphagia, dysarthria, oculogyric crisis); responds well to benztropine or diphenhydramine
 - (4) Tardive dyskinesia (sucking, lip-smacking, perioral "rabbit" syndrome): appears late, after prolonged therapy.

- (5) Newer second-generation antipsychotics (eg, clozapine, risperidone, olanzapine) exhibit less dopamine₂-receptor blockade and have a lower rate of extrapyramidal adverse effects.
- b. Orthostatic hypotension
- c. Hypo- or hyperthermia
- d. Neuroleptic malignant syndrome
- 3. Acute overdose
 - a. Clinical presentation
 - (1) Anticholinergic
 - (2) α -adrenergic blockade (miosis, vasodilation, hypotension, "warm shock")
 - (3) Sodium/potassium channel blockade and cardiodepressant effects are most common with thioridazine and mesoridazine.
 - (a) Prolonged PR, QRS, and QT intervals
 - (b) ST and T wave changes
 - (c) Ventricular dysrhythmias (including torsades de pointes) and AV dissociation
 - (4) Coma, rigidity, and seizures
 - b. Management (conservative)
 - (1) Activated charcoal
 - (2) Dialysis is not useful.
 - (3) Respiratory compromise → airway management
 - (4) Hypotension → IV fluids
 - (5) Seizures → diazepam, lorazepam, phenobarbital
 - (6) Extrapyramidal reactions → benztropine or diphenhydramine
 - (7) Ventricular dysrhythmias
 - (a) NaHCO₃ should be tried first (see cyclic antidepressants).
 - (b) Magnesium is indicated for torsades de pointes.
 - (c) Do not use procainamide, quinidine, or other class IA or IC antidysrhythmics.

F. Phenytoin

- 1. Pathophysiology
 - a. Correlation of blood levels with evolution of clinical effects
 - (1) 10–20 mcg/mL → therapeutic level
 - (2) 20 mcg/mL → lateral gaze nystagmus
 - (3) 20–30 mcg/mL → nystagmus and cerebellar excitation
 - (4) 30–40 mcg/mL → nausea, vomiting, ataxia, dysarthria
 - (5) >40 mcg/mL → confusion and lethargy
 - (6) 50 mcg/mL → choreoathetosis, opisthotonus (rarely)
 - (7) >100 mcg/mL → potentially lethal level
 - b. Cardiac actions
 - (1) Decreased automaticity
 - (2) Cardiac toxicity, especially ventricular dysrhythmias
 - (a) May occur with IV loading of phenytoin (commonly attributed to too rapid infusion of the propylene glycol diluent)

- (b) Fosphenytoin is a water-soluble phenytoin pro-drug for parenteral use and does not contain propylene glycol; cardiac toxicity much more rare with fosphenytoin but has been reported.
- c. Miscellaneous effects
 - (1) Osteomalacia (chronic use)
 - (2) Gingival hypertrophy (chronic use)
 - (3) Megaloblastic anemia
 - (4) Fetal hydantoin syndrome
 - (5) Skin rashes and the anticonvulsant hypersensitivity syndrome (fever, rash, lymphadenopathy, eosinophilia), which will recur if phenobarbital or carbamazepine are substituted
- 2. Management of acute ingestion
 - a. Repeated doses of activated charcoal may be useful. Use with caution, if at all, in patients dependent on phenobarbital (eg, those with alcohol dependence or seizure disorders). In these patients, supportive measures may be best.
 - b. Supportive care until confusion and ataxia clear.

G. Opioids

- 1. Classic clinical scenarios
 - a. Opioid withdrawal
 - (1) Clinical presentation: abdominal cramping/pain, chills, sweating, nausea, diarrhea, and arthralgias; distinctive physical findings include yawning, lacrimation, rhinorrhea, and piloerection.
 - (2) Onset is usually within 12–24 hours of the last dose but is longer with sustained-release opioids and methadone.
 - (3) Symptoms peak 1–2 days after onset and subside within a week.
 - b. Opioid intoxication and overdose
 - (1) Clinical presentation: euphoria, drowsiness, decreased respiratory rate, miosis
 - (2) Severe overdoses can cause coma and respiratory acidosis.
 - (3) Seizures may occur after tramadol, propoxyphene, or meperidine overdose.
- 2. Specific treatment of opioid overdose → naloxone
 - a. Initial dose is 0.1–2 mg in adults and children and should be titrated carefully. It may be given IV, IM, SC, endotracheally, intranasally. It is effective within minutes. Higher doses may be needed to reverse the following agents: buprenorphine, propoxyphene, pentazocine, meperidine, fentanyl, and diphenoxylate.
 - b. It should be used to reverse respiratory depression and dosed until respiratory depression has resolved. *Note:* Naloxone will precipitate acute withdrawal in opioid-dependent patients, which is the major complication of its use.
 - c. The effect of naloxone decreases 15–30 minutes after injection, but the action of the opioid may last for hours.
 - (1) Buprenorphine, methadone, controlled-release opioids, and diphenoxylate/atropine in particular last >12–24 hours.
 - (2) Repeated doses and prolonged observation may be required.
 - (3) Patients who require naloxone after buprenorphine, methadone, sustained-release opioid, or diphenoxylate/atropine overdose need admission.

- (4) Patients may need a continuous infusion of naloxone. Titrate based on respiratory depression versus signs of withdrawal.
3. Acute complications of opioid abuse
 - a. Seizures (meperidine, propoxyphene, tramadol, pentazocine, dextromethorphan)
 - b. Pulmonary edema
 - c. Rhabdomyolysis
 - d. Aspiration pneumonia

H. Clonidine

1. Pathophysiology
 - a. A centrally acting α_2 -agonist, clonidine reduces sympathetic stimulation to the heart and peripheral blood vessels.
 - b. The initial effect is hypertension followed by an antihypertensive effect that occurs up to 10 hours later.
 - c. Withdrawal is associated with severe headache, rebound hypertension, anxiety, and tremors.
2. Overdose
 - a. Clinical presentation
 - (1) Mimics opioid overdose and includes apnea, altered level of consciousness (drowsiness \rightarrow coma), seizures, and miosis; hypotension and bradycardia are prominent features.
 - (2) Children are more severely affected than adults.
 - b. Management
 - (1) Supportive
 - (2) Respiratory support, atropine, vasopressors, and GI decontamination
 - (3) Naloxone has been used with variable success to reverse CNS and respiratory depression associated with clonidine poisoning. High doses may be needed (10–20 mg IV).

I. Alcohols

1. Methanol

- a. Sources and pathophysiology
 - (1) Found in windshield washing fluids, solvents, paint thinners, and canned fuels. Suspect multiple victims if ingestion was recreational substitution for ethanol.
 - (2) Converted by alcohol dehydrogenase to formaldehyde, which is then converted to formic acid.
 - (a) The accumulation of formic acid correlates with the decrease in bicarbonate, the increase in anion gap, and the severity of metabolic acidosis.
 - (b) Formic acid affects optic nerve function, producing optic papillitis and retinal edema \rightarrow "blind drunk"
- b. Classic clinical scenario: A latent period of 8–30 hours is followed by the onset of abdominal pain, nausea and vomiting, blurred vision, and metabolic acidosis. The patient may have normal vision (and a normal eye examination) initially. As the formic acid accumulates, visual symptoms include photophobia, "snowstorm" vision, and blindness. The pupils dilate and react sluggishly, if at all, to light. The earliest funduscopic finding is hyperemia of the disc, followed by papilledema and optic atrophy. Late parkinsonism may result.

c. Diagnostic evaluation

- (1) Anion gap metabolic acidosis associated with a low bicarbonate level
- (2) The osmol gap may be high, although a low or even "negative" gap does not exclude a toxic alcohol or glycol.
 - (a) Osmolar gap: calculated osmolality – measured osmolality
 - (b) Calculated osmolality

$$(2 \times \text{Na}) + \frac{\text{BUN}}{2.8} + \frac{\text{Glucose}}{18} + \frac{\text{Ethanol}}{4.6}$$

- (c) Normal serum osmolality: 280–295 mOsm
- (d) Osmotically active substances that may produce an osmol gap
 - i. Methanol
 - ii. Ethanol
 - iii. Isopropyl alcohol
 - iv. Glycerol
 - v. Mannitol
 - vi. Ethylene glycol
- (e) Osmol gaps can also be seen in lactic acidosis and alcoholic ketoacidosis, or with high serum ethanol concentrations (despite "correction" using the above formula).

d. Management

- (1) General supportive measures, including airway management
- (2) Nasogastric aspiration of gastric contents if the patient is seen within the first 30–60 minutes after ingestion
- (3) Severe acidosis is treated with bicarbonate to reduce diffusion of formate into the CNS and protect the optic nerve.
- (4) Alcohol dehydrogenase inhibitor to block further metabolism of methanol.
 - (a) Indications: methanol level >20 mg/dL (may start empirically while waiting for level)
 - (b) Fomepizole (4-methylpyrazole) is the preferred agent.
 - (c) Second-line agent: ethanol
 - i. Difficult to find and to dose
 - ii. Goal blood ethanol level: 100–150 mg/dL
- (5) Hemodialysis removes methanol and formic acid and is indicated for methanol level >50 mg/dL, metabolic acidosis (arterial pH ≤7.25), severe visual or CNS symptoms, or when ethanol is used for therapy.
- (6) Folinic acid can be considered and may enhance metabolism of formic acid to nontoxic substances, which are then eliminated from the body.

2. Ethylene glycol

a. Source and pathophysiology

- (1) Found in antifreeze and brake fluids
- (2) Converted by alcohol dehydrogenase to glycolaldehyde, which is then metabolized to glycolic acid.

- (a) Glycolic acid is primarily responsible for the anion gap metabolic acidosis; it is ultimately metabolized to multiple metabolites, including oxalic acid.
 - (b) Oxalic acid forms calcium oxalate crystals in the kidney, brain, and liver.
 - b. Clinical presentation
 - (1) CNS changes suggesting ethanol intoxication
 - (2) Progressive toxicity over 9–12 hours can lead to seizures, stupor, and coma.
 - (3) As toxicity progresses, pulmonary edema and myocardial dysfunction can develop; death may occur.
 - (4) Late toxicity is characterized by renal failure.
 - (5) Cranial nerve (frequently facial) dysfunction can develop (late finding).
 - c. Diagnostic evaluation
 - (1) Anion gap metabolic acidosis
 - (2) Positive birefringent calcium oxalate crystals in the urine
 - (a) Often absent initially
 - (b) Commonly described as “envelope-shaped”
 - (3) Urine that fluoresces under a Wood’s lamp may be evident if the patient recently ingested a fluorescein-containing antifreeze; however, this has been shown to be very unreliable, and false-positives and false-negatives are common.
 - (4) Osmol gap (absence does not exclude ingestion)
 - (5) Hypocalcemia (not always present)
 - d. Management
 - (1) Gastric decontamination, IV ethanol or fomepizole, and hemodialysis (same as for methanol toxicity)
 - (2) Thiamine and pyridoxine can be administered to decrease production of oxalic acid.
 - (3) Calcium administration as needed (long QT interval)
3. Ethanol
- a. Intoxication
 - (1) Degree of intoxication varies according to the patient’s tolerance.
 - (2) Clinical presentation
 - (a) Mild intoxication (euphoria, expansiveness, loss of self-control)
 - (b) Moderate intoxication
 - i. Slurred speech, ataxia, and nystagmus
 - ii. Altered sensory perceptions that are dull, distorted, or obviously psychotic
 - iii. Mild tachycardia is common.
 - (c) Severe intoxication (the four “H’s”)
 - i. Hypotension
 - ii. Hypoventilation
 - iii. Hypothermia
 - iv. Hypoglycemia (more common in small children)
 - (3) Care must be taken to consider coexisting disease, hypoglycemia, trauma (especially closed head injury), or drug ingestion.

(4) Management

- (a) Most patients with uncomplicated ethanol intoxication require only observation and basic supportive measures.
- (b) Gastric decontamination is indicated if polydrug ingestion is known or suspected.
- (c) Administer IV glucose, naloxone, and thiamine 100 mg, when necessary.
- (d) Hemodialysis is indicated only for life-threatening complications (refractory hypotension).

b. Withdrawal**(1) Clinical presentation**

- (a) Early signs and symptoms: tremor, irritability, increased pulse, increased blood pressure
- (b) Delirium tremens (onset is 48–100 hours after cessation)
 - i. Hypertension, tachycardia
 - ii. Diaphoresis, dehydration
 - iii. Visual hallucinations and paranoid ideation
- (c) Withdrawal seizures ("rum fits")
 - i. Generally occur within 6–48 hours of last drink
 - ii. Usually single or several brief generalized seizures with rapid recovery in between
 - iii. Intracranial pathology must be considered in the presence of fever, prolonged postictal period, signs of trauma, or focality.
- (d) Wernicke-Korsakoff syndrome (thiamine deficiency)—two of the following signs:
 - i. Dietary deficiencies
 - ii. Cerebellar dysfunction
 - iii. Oculomotor abnormalities
 - iv. Altered mental state or mild memory impairment

(2) Management

- (a) IV fluids, thiamine, and glucose
- (b) Replacement of potassium, magnesium, and phosphorus when indicated
- (c) Benzodiazepines for control of the withdrawal state
 - i. Enough medication should be administered to calm or lightly sedate the patient. For example, diazepam can be started at 5–10 mg, then doubled until desired effect; very large doses may be needed.
 - ii. Phenytoin is not effective for alcohol-withdrawal seizures.
 - iii. Physical restraints should be used to temporarily control patients only until the pharmacologic treatment becomes effective. A patient should never be allowed to thrash uncontrollably in physical restraints, unattended, for a prolonged time.
 - iv. Avoid antipsychotics (haloperidol, ziprasidone): may lower seizure threshold.
 - v. Sedation with propofol or barbiturates may be necessary in severe refractory cases.

4. Isopropanol

a. Pathophysiology

- (1) Isopropyl (rubbing) alcohol causes more intoxication/CNS depression but generally less severe sequelae than methanol and ethylene glycol.
- (2) 80% is absorbed from the stomach within 30 minutes and is then metabolized (by alcohol dehydrogenase) to acetone.

b. Clinical presentation

- (1) CNS depression
- (2) Hypotension (in severe cases)
- (3) Hemorrhagic gastritis/tracheobronchitis

c. Diagnostic evaluation

- (1) Positive serum acetone and acetonuria
- (2) High osmolal gap
- (3) Anion gap metabolic acidosis is not a feature of isopropanol toxicity (unless hypotension with lactic acidosis is present). This finding should prompt a search for another cause (eg, sepsis, alcoholic ketoacidosis, ethylene glycol, or methanol coingestion).

d. Management

- (1) Supportive care, including airway management
- (2) Nasogastric aspiration of gastric contents within initial 30–60 minutes
- (3) Alcohol dehydrogenase inhibitors (fomepizole, ethanol) are not indicated.
- (4) Search for any complication or concomitant condition (eg, trauma)
- (5) Indications for hemodialysis
 - (a) Refractory hypotension
 - (b) Serum levels >400–500 mg/dL

J. Cocaine

1. Pathophysiology

- a. Metabolized by the liver, plasma esterases, and spontaneous hydrolysis within a few hours, then excreted in the urine
- b. Causes euphoria with lack of fatigue, a sense of omnipotence, increased mental alertness, and sexual stimulation

2. Clinical presentation

- a. CNS stimulation → apprehension, twitching, seizures; coma can develop later.
- b. Cardiac stimulation
 - (1) Hypertension, tachycardia, ventricular dysrhythmias, cardiac conduction blocks
 - (2) Effects on coronary arteries and increased platelet aggregation can lead to an acute MI; this finding in a young person should raise the suspicion of cocaine use.
- c. Respiratory depression/failure
 - (1) Pulmonary edema
 - (2) Pneumothorax/mediastinal air
 - (3) Crack lung (bronchospasm, infiltrates, eosinophilia hemorrhage)
- d. Hyperthermia (may occur suddenly and can be lethal)

- e. Rhabdomyolysis → hyperkalemia (can be life-threatening)
 - f. Arterial vasospasm can lead to tissue infarction in various tissues, such as brain, lung, liver, and intestines. Intracerebral hemorrhages and aortic dissection can also occur.
3. Cocaine-associated chest pain → acute MI in 6% of patients
- a. Abnormal ECG is common (early repolarization, old and new nonspecific abnormalities)
 - b. Management
 - (1) Benzodiazepines are first-line treatment for hypertension and tachycardia.
 - (2) Pharmacologic agents to control pulse rate and blood pressure (eg, phentolamine), along with benzodiazepines, should be started.
 - (3) Angioplasty is preferred over systemic fibrinolysis for persistent ST-segment elevation MI despite the above measures.
 - (4) Patients without ischemic changes and negative cardiac markers over an appropriate observation period can usually be managed as outpatients.
4. Management
- a. General supportive measures
 - (1) Airway management and IV hydration to assure a urinary output of 1–2 mL/kg/hr.
 - (2) Cardiac monitoring and repeat vital signs (including temperature) every few minutes.
 - b. Seizures, agitation, and psychosis are best managed with benzodiazepines. Phenothiazines (eg, chlorpromazine) and haloperidol may lower the seizure threshold and prevent heat dissipation in the already hyperthermic patient; avoid when treating cocaine-induced psychosis.
 - c. Cocaine-induced cardiac dysrhythmias should be treated with IV benzodiazepines. Sodium bicarbonate should be considered for dysrhythmias refractory to benzodiazepines (especially those with a wide QRS complex). Drugs that should be avoided include:
 - (1) Lidocaine (may increase CNS toxicity → seizures)
 - (2) β -blockers (may precipitate cardiac ischemia or hypertension due to unopposed α -receptor stimulation)
 - (3) α -blockers (eg, phentolamine) followed by use of a β -blocker (or combination agents, such as labetalol) have been used in these patients to alleviate concerns for unopposed α -receptor stimulation and subsequent hypertensive crisis.
 - d. Hyperthermia should be treated in the usual fashion. Aggressive measures may be needed. See pages 826–827.
 - e. Dialysis has no role in the management of cocaine poisoning.
 - f. Body stuffers and body packers—specific management:
 - (1) Activated charcoal for body stuffers
 - (2) Whole-bowel irrigation for body packers
 - (3) Observation
 - (a) Body stuffers (nonprofessionally wrapped packets, eg, impromptu swallowing of baggies to avoid arrest) → until asymptomatic (6–12 hours)
 - (b) Body packers (professionals) → until all packets have passed (may need to be confirmed with radiocontrast studies)

K. Amphetamines/amphetamine-like drugs

1. Commonly abused drugs
 - a. Ephedrine (ma huang, herbal ecstasy)
 - b. Methamphetamine (crank, ice)—can also be smoked
 - c. MDMA (Ecstasy), MDEA (Eve), etc
 - d. Cathinone (Khat)
 - e. Synthetic cathinones ("bath salts")
2. Pathophysiology
 - a. Rapidly absorbed from GI tract
 - b. Eliminated via liver (30%–45%) and kidneys (60%–70%)
3. Clinical presentation
 - a. Prescription doses
 - (1) Central effects (anorexia, hyperactivity, restlessness, and sleeplessness)
 - (2) Peripheral effects (tachycardia, vasoconstriction, bronchodilation, and pupillary dilation)
 - (3) Phenylpropanolamine was removed from OTC diet and decongestant preparations because of concerns with hypertension, seizures, intracerebral hemorrhage, and dysrhythmias.
 - b. Withdrawal signs and symptoms
 - (1) Headache
 - (2) Increased appetite
 - (3) Abdominal cramps and diarrhea
 - c. Toxic signs and symptoms can be clinically indistinguishable from those of cocaine toxicity.
 - (1) Cardiovascular: hypertension, tachycardia, dysrhythmias, MI, intracerebral hemorrhage
 - (2) Neurologic: diaphoresis, mydriasis, piloerection, extreme restlessness, psychotic behavior, seizures, coma
 - (3) GI: nausea, vomiting, diarrhea
 - (4) Other toxic signs
 - (a) Hyperthermia
 - (b) Rhabdomyolysis
 - (c) Hallucinations (occur with designer amphetamines, eg, MDMA, MDEA)
 - (d) Hyponatremia (due to syndrome of inappropriate antidiuretic hormone secretion with MDMA)
4. Management
 - a. General measures
 - (1) Quiet area to reduce hyperactivity
 - (2) Cardiac monitor
 - (3) Aggressive cooling measures for hyperthermia
 - (4) Activated charcoal for ingestions
 - (5) Correction of fluid and electrolyte disturbances

b. Specific therapy

- (1) Benzodiazepines for seizures and agitated psychosis
- (2) Nitroprusside, phentolamine, or labetalol for hypertension unresponsive to benzodiazepine sedation (avoid β -blockers)

L. Hallucinogens

1. Phencyclidine (PCP)

a. Clinical presentation

- (1) Most common findings are abnormal vital signs (hypertension, tachycardia, hyperthermia) and nystagmus (horizontal, vertical, or rotary) in a patient with bizarre behavior that can fluctuate between severe agitation and catatonic-like behavior.
- (2) Pupillary size is variable, but miosis is common.
- (3) Severe hyperthermia, seizures, coma, and rhabdomyolysis can occur with large ingestions.

b. Management

- (1) Benzodiazepines for agitation and seizures
- (2) Aggressive cooling measures for severe hyperthermia
- (3) Hydrate with IV crystalloids to maintain a urinary output of 1–2 mL/kg/hr
- (4) Activated charcoal should be given; repeated doses may be efficacious because of the gastroenteric circulation of PCP.
- (5) Acidification of the urine is no longer recommended.

2. Other hallucinogens

- a. LSD (psychedelic flashbacks are a prominent feature, especially in chronic users)
- b. Mescaline
- c. Psilocybin (see mushroom poisoning, p 734)
- d. Cannabinoids
- e. Jimsonweed (*Datura*) (anticholinergic toxicity)
- f. Morning glory plant (*Ipomoea*) (anticholinergic toxicity)
- g. Nutmeg (large doses)
- h. Ketamine (vitamin K)
- i. Bufotenine (toad licking)
- j. Yohimbine
- k. Designer amphetamines (MDMA, MDEA, DOM/STP, "bath salts")

3. General treatment of hallucinogens

- a. A quiet, semidarkened room
- b. Benzodiazepines for sedation (and seizures)
- c. Temperature monitoring and aggressive cooling for severe hyperthermia.

M. Salicylates (includes ASA, oil of wintergreen, bismuth subsalicylate, liniments)

1. Acid-base disorders occur in three phases

a. First phase: primary respiratory alkalosis

- (1) Salicylate stimulates the respiratory center in cerebral medulla → respiratory alkalosis
- (2) May not be seen in children

- b. Second phase: primary metabolic acidosis, blood pH remains normal
 - (1) Primary respiratory alkalosis also serves as compensation for this developing primary metabolic acidosis.
 - (2) Salicylate uncouples oxidative phosphorylation, leading to anion gap metabolic acidosis.
 - c. Third phase: overwhelming metabolic acidosis
 - (1) Patient can no longer compensate for metabolic acidosis.
 - (2) Acidemia and shock ensue.
2. Acute versus chronic poisoning
- a. Acute ingestion
 - (1) Mild toxicity (triad)
 - (a) Ototoxicity (tinnitus, vertigo, and hearing distortion)
 - (b) Tachypnea and hyperpnea
 - (c) Nausea and vomiting, abdominal pain
 - (2) Severe toxicity
 - (a) CNS signs and symptoms dominate the clinical picture with signs of stimulation occurring initially, followed by CNS depression (lethargy or excitability, confusion, delirium, psychosis → stupor, convulsions, coma).
 - (b) Definitive signs of severe toxicity
 - i. Cardiac dysrhythmias
 - ii. Noncardiogenic pulmonary edema
 - iii. Acute renal failure
 - iv. Hemorrhage
 - v. Hypo- or hyperglycemia
 - vi. Ketonemia, ketonuria
 - (c) Other associated signs and symptoms
 - i. Hyperthermia
 - ii. Diaphoresis
 - iii. Dehydration (especially in children)
 - b. Chronic ingestion (most commonly seen in the elderly)
 - (1) Suspect in any patient with unexplained CNS dysfunction, especially in the presence of a mixed acid-base disturbance
 - (a) Change in mentation is the usual presenting complaint (confusion, disorientation, lethargy, or hallucinations).
 - (b) Respiratory alkalosis with anion gap metabolic acidosis and a normal or increased pH is the most frequent presenting acid-base abnormality.
 - (2) Noncardiogenic pulmonary edema
3. Diagnostic evaluation
- a. Serum salicylate: a serum ASA level should be drawn at presentation and repeated every 4 hours until the serum level is clearly trending down and below 20 mg/dL. The Done nomogram has little usefulness in assessing salicylate toxicity.
 - b. Blood gases (venous or arterial) determine the type and degree of acid-base imbalance.

- c. Chemistries
 - (1) Electrolytes → exclude ↓ K^+ , ↓ Na^+ , ↓ HCO_3^-
 - (2) Glucose → exclude hypoglycemia, hyperglycemia; in salicylate-poisoned patients, CSF may be hypoglycemic despite a normal serum glucose.
 - (3) BUN/creatinine → exclude renal failure
- d. Urinary ferric chloride testing is littered with false-positives and false-negatives and cannot be reliably used to exclude salicylate poisoning.
- 4. Management
 - a. GI contamination: consider activated charcoal. Multiple doses of charcoal given every 2–4 hours are recommended by some but have not been shown to be beneficial. Gastric lavage may be considered.
 - b. Urine alkalinization
 - (1) Renal excretion of salicylate (an acid) is enhanced by ionization; urinary excretion of salicylates increases 10- to 20-fold when urine pH is increased from 5 to 8.
 - (2) Urinary alkalinization can be accomplished by bolus and drip.
 - (a) Bolus: 1–2 mEq/kg $NaHCO_3$
 - (b) Drip: 150 mEq $NaHCO_3$ in 850 mL D5W + 40 mEq KCl
 - i. Start at 1.5–2 times the maintenance fluid rate and adjust as needed to maintain a urine pH of 7–8 without exceeding a serum pH >7.5.
 - ii. Potassium replacement is necessary, because hypokalemia prevents urinary alkalinization due to exchange of H^+ for K^+ in the renal tubules.
 - (3) Indications
 - (a) Proven salicylate toxicity with symptoms
 - i. Caution: symptoms may be subtle; a thorough history and physical examination are essential.
 - ii. Check specifically for tinnitus, hearing changes, and hyperventilation.
 - (b) Suspected toxicity (initiate before confirmatory laboratory results): tinnitus, delirium, hyperthermia, metabolic derangements
 - (4) Contraindications
 - (a) Benzodiazepines for agitation → respiratory drive → serum pH → shift of salicylic acid into CNS → rapid deterioration
 - (b) Urinary alkalinization with acetazolamide → worsening of systemic acidosis → ↑ ASA in CNS
 - c. Hemodialysis is the most effective means of lowering the serum salicylate level and is indicated when one or more of the following conditions is present:
 - (1) Serum ASA level >90–100 mg/dL in any patient regardless of symptoms. Some recommend hemodialysis in chronic salicylate poisoning when levels exceed 40–60 mg/dL.
 - (2) Neurologic signs and symptoms (confusion, delirium, psychosis, stupor, coma, or seizures)
 - (3) Renal or hepatic failure
 - (4) Pulmonary edema
 - (5) Severe cardiac toxicity
 - (6) Severe acid-base imbalance
 - (7) Rising serum ASA levels despite urinary alkalinization

5. Complications of salicylate toxicity
 - a. Gastric dilation and ileus
 - b. GI hemorrhage
 - c. Platelet dysfunction

N. Acetaminophen

1. Phases of acetaminophen poisoning
 - a. Phase I—30 minutes to 24 hours after ingestion: anorexia, nausea, and vomiting; pallor and diaphoresis may also be present
 - b. Phase II—24–72 hours after ingestion: increased hepatic enzymes, INR, and bilirubin; right upper quadrant pain may be present
 - c. Phase III—72–96 hours after ingestion: hepatic encephalopathy, coagulation defects, jaundice and renal failure; myocardial dysfunction may be present; anuria and coma are ominous signs
 - d. Phase IV—4 days to 2 weeks after ingestion: if the pathologic effects of Phase III are reversible, hepatic dysfunction will completely resolve.
2. Evaluation of toxicity
 - a. Toxicity may occur with ingestions >200 mg/kg.
 - b. The Rumack Matthew nomogram is used to determine the need for treatment after a single, acute ingestion.
 - (1) The first serum acetaminophen level should be drawn no earlier than 4 hours after ingestion. Only one level is required to determine the need for treatment.
 - (2) If the measured concentration falls above the lower line on the nomogram, treatment with N-acetylcysteine (NAC) should be started.
 - (3) If the level falls below the lower line, no treatment is needed.
 - (4) IV and oral NAC are equally effective.
3. Management
 - a. Activated charcoal should be considered for patients presenting within 1 hour of a potentially toxic ingestion (200 mg/kg). Simultaneous activated charcoal and NAC is unlikely to cause consequential decrease in NAC availability (as was previously thought).
 - b. **N-acetylcysteine therapy is most efficacious when started within 8 hours of ingestion; however, therapy begun after 8 hours retains a high margin of efficacy and should therefore still be given to late-presenting patients.**
 - (1) Mechanism of action
 - (a) Acetaminophen is converted to an intermediate reactive metabolite by the cytochrome p-450 system. This metabolite (NAPQI) is normally detoxified by glutathione in the liver.
 - (b) Excess acetaminophen depletes glutathione stores and the toxic metabolite damages hepatic cells, causing centrilobular hepatic necrosis.
 - (c) The exact mechanism by which NAC prevents hepatotoxicity is uncertain and likely multifactorial. Possible mechanisms include the following:
 - i. May act by increasing synthesis of glutathione
 - ii. Functions as a glutathione substitute, combining directly with the toxic metabolite

- iii. Enhances the sulfonation pathway of acetaminophen metabolism (nontoxic)
- iv. Serves as a free-radical scavenger
- v. Converts NAPQI back to acetaminophen

(2) Dosage schedule

- (a) Oral (72-hour) regimen
- (b) 20-hour IV NAC: sick patients, usually those who present late after an acetaminophen overdose, may require extension of the protocol beyond 20 hours. If an anaphylactoid reaction occurs during the initial load, stop the infusion and administer diphenhydramine; then reassess the need for NAC therapy and, if indicated, resume at a slower rate. This will resolve most anaphylactoid reactions; however, should it recur, the patient can be switched to the oral regimen. Anaphylactoid reactions are not reported with the oral regimen.
- c. NAC is also indicated for already developed acetaminophen hepatotoxicity (late-presenting patients). The mechanisms by which NAC may ameliorate hepatotoxicity in these patients include:
 - (1) Free-radical scavenger
 - (2) Improved hepatic oxygen delivery and consumption; NAC therapy should continue in all hepatotoxic patients until recovery (patient is awake, serum acetaminophen undetectable, AST or ALT falling, and INR <2.0).
- d. Patients with the following criteria (King's College Criteria) should be referred to a toxicology treatment/liver unit:
 - (1) pH <7.3 after fluid resuscitation or
 - (2) All three of:
 - (a) Prothrombin time >100 seconds (INR >6.5 has been used)
 - (b) Creatinine > 3.3 mg/dL
 - (c) Grade III or IV encephalopathy
 - (3) Note the absence of hepatic transaminases in the King's College Criteria; they are not useful for determining prognosis after acetaminophen poisoning.
- e. MELD criteria are less predictive of the need for transplant in the acetaminophen-poisoned patient and are therefore not preferred for risk assessment in these patients.

O. Iron

1. Pathophysiology and toxicity

- a. Iron is absorbed in the duodenum and upper small bowel and enters the plasma, where it is bound by transferrin. When transferrin sites are saturated, free iron is available to produce tissue damage. A high dose causes GI necrosis with subsequent hemorrhage.
- b. Toxic ingestions are determined by the amount of elemental iron contained in the tablet and the number of tablets ingested.
 - (1) Iron preparations
 - (a) Ferrous sulfate (20% elemental iron): for example, a 300-mg tablet contains 60 mg of elemental iron.
 - (b) Ferrous fumarate (33% elemental iron)
 - (c) Ferrous gluconate (12% elemental iron)
 - (2) A potentially toxic ingestion of elemental iron is 20 mg/kg. More than 5 tablets of any iron preparation may be a toxic ingestion for a child.

2. Clinical stages of iron poisoning (in severe poisoning, stages may overlap)
 - a. Stage I: 30 minutes to 6 hours after ingestion: emesis, diarrhea, abdominal pain, hematemesis, and hematochezia; conversely, the absence of GI symptoms for 6 hours after ingestion excludes severe toxicity.
 - b. Stage II: 4–12 hours after ingestion: GI symptoms improve, although patient status is not normal; subclinical hypoperfusion and metabolic acidosis manifest. Assess carefully for signs of early hypoperfusion and monitor arterial blood gases.
 - c. Stage III: 6–72 hours after ingestion: coma, metabolic acidosis, coagulation problems, shock, and seizures
 - d. Stage IV: 12–96 hours after ingestion: hepatic failure with jaundice, hypoglycemia, and coagulopathy
 - e. Stage V: 2–4 weeks after ingestion: vomiting, abdominal pain and pyloric scarring, gastric outlet and small-bowel obstruction
3. Diagnostic evaluation
 - a. Serum iron concentration
 - (1) A level >350 mcg/dL 3–5 hours after ingestion may indicate the need for chelation therapy.
 - (2) Repeat levels should be obtained to be sure they are not rising.
 - (3) Total iron binding capacity is not reliable in this setting, as was previously thought.
 - b. Other studies
 - (1) CBC, coagulation profile, glucose, electrolytes, and BUN
 - (2) Blood gases are indicated if the patient is symptomatic.
 - (3) Blood glucose and a WBC count, formerly thought to be predictive of serum iron levels >300 mcg/dL, are now known to be insensitive.
 - c. An abdominal radiograph can identify undissolved tablets but is not helpful if negative.
4. Management
 - a. Decontamination
 - (1) Gastric emptying
 - (2) Whole-bowel irrigation is indicated for a large ingestion, especially if radiopaque tablets are seen on abdominal radiograph.
 - b. IV fluids are indicated if the patient is hypotensive or has severe gastroenteritis.
 - c. Deferoxamine therapy
 - (1) Indications
 - (a) Serum iron concentration >350 mcg/dL and the patient has symptoms (including protracted vomiting)
 - (b) Shock
 - (c) Coma or seizures
 - (d) Acidosis
 - (2) Administration: 15 mg/kg/hr continuous IV infusion to a maximum of 6 g; hypotension is the main adverse effect.
 - (3) The iron-deferoxamine complex is excreted in the urine and turns it an orange-red color (classically referred to as “vin rose”). Color changes can be subtle, and the color difference should not be used to guide therapy.
 - (4) The deferoxamine challenge is no longer used diagnostically or therapeutically.

P. Hydrocarbons

1. Definitions

- a. Hydrocarbons are a broad group of organic compounds that contain carbon and hydrogen in various configurations. The halogenated hydrocarbons also contain halogens such as chlorine or fluorine.
- b. Petroleum distillates are hydrocarbons, but not all hydrocarbons are petroleum distillates. Petroleum distillates are the breakdown products remaining after processing crude oil. Hydrocarbons can also be derived from coal or plant sources.

2. **The aspirational hazard of a specific hydrocarbon depends on whether or not it has two physical properties: low viscosity and high volatility. Viscosity is by far the most important factor.**

3. Clinical presentation of hydrocarbon ingestion

- a. Signs and symptoms of aspiration include coughing, choking, and gagging. Cyanosis may be evident within minutes.
- b. Other signs
 - (1) CNS (lethargy, seizures) → camphor, lindane, eucalyptus oil
 - (2) Respiratory (wheezing, rales, rhonchi, decreased breath sounds, pulmonary edema); radiographic findings do not always correlate with physical signs.
 - (3) Miscellaneous (tachycardia, fever, belching, vomiting, diarrhea)

4. Management

a. Asymptomatic patient

- (1) Indications for gastric aspiration (nasogastric tube)
 - (a) Presence of a toxic, or "CHAMP," substance within the ingested agent:
 - Camphor
 - Halogenated hydrocarbons
 - Aromatics
 - Metals
 - Pesticides
 - (b) Ingestion of a massive amount of a low-viscosity agent
- (2) Chest radiograph is controversial and generally not useful in asymptomatic, well-appearing patients.
- (3) Disposition
 - (a) Children: observe and reassess for 6 hours in the emergency department.
 - (b) Adults (responsible): discharge with specific instructions to return if symptoms develop.

b. Symptomatic patient

- (1) Chest radiograph
- (2) Supportive care and airway management
- (3) Specific therapy
 - (a) Bronchospasm → nebulized β -agonists
 - (b) Pulmonary edema → continuous positive-airway pressure/positive end-expiratory pressure

- (4) Precautions
 - (a) **Steroids are not useful.**
 - (b) **Antibiotics should be used only for definite infection.**
- 5. Aromatics (solvents)
 - a. Most exposures occur through inhalation, because aromatics evaporate readily at room temperature.
 - b. Clinical presentation
 - (1) Benzene
 - (a) Acute exposure causes CNS depression.
 - (b) Chronic exposure causes bone marrow aplasia, leukemia.
 - (2) Toluene, xylene
 - (a) Respiratory tract irritation
 - (b) A state of inebriation that can progress to CNS depression
 - (c) Renal tubular acidosis (toluene)
 - c. Management is symptomatic and supportive. The use of epinephrine for bronchospasm should be avoided, because aromatics sensitize the myocardium.
- 6. Halogenated hydrocarbons (toxicity is by inhalation, and treatment is supportive and symptomatic) sensitize the myocardium, thus predisposing the patient to dysrhythmias. Signs and symptoms that differentiate specific agents are listed below.
 - a. Carbon tetrachloride (tetrachloromethane)
 - (1) Causes marked hepatic and renal toxicity
 - (2) Clinical presentation
 - (a) Immediate effects → nausea, vomiting, diarrhea, and abdominal pain
 - (b) Exposure to high concentrations → dizziness, confusion, coma, respiratory depression, and hypotension
 - (c) Prolonged exposure → polyneuritis, visual disturbances, and anemia
 - (3) In addition to supportive and symptomatic treatment, NAC and hyperbaric oxygen therapy are also used for serious poisoning.
 - b. Chloroform (trichloromethane)
 - (1) Found in cleaning solvents and aerosol propellants
 - (2) CNS signs and symptoms, plus skin and eye irritation, dominate the clinical picture.
 - (3) The patient may "appear drunk," but his or her breath smells of chloroform, not alcohol.
 - c. Methylene chloride
 - (1) Found in paint remover, degreasing solvents, aerosol solvents/propellants, and refrigerants.
 - (2) On inhalation, pulmonary and CNS signs and symptoms dominate the clinical picture.
 - (3) Methylene chloride is partially metabolized to carbon monoxide, so carboxyhemoglobin levels should be monitored.
 - (a) Patients may develop toxic carboxyhemoglobin levels after inhalation or ingestion of methylene chloride.
 - (b) Peak levels may not be seen for ~8 hours.

d. Essential oils

- (1) Found in popular home remedies, liniments, and "aromatherapy." All have CNS effects and cause oral numbness/irritation and GI distress when ingested.
- (2) Specific toxicities
 - (a) Camphor → rapid onset of seizures
 - (b) Nutmeg → hallucinations
 - (c) Eucalyptus oil → seizures, coma
 - (d) Pennyroyal oil → acetaminophen-like hepatotoxicity

Q. Caustic ingestions

1. Pathophysiology

- a. Alkali ingestion (eg, lye or sodium hydroxide)
 - (1) The esophagus is injured more often than the stomach.
 - (2) Injury results from liquefaction necrosis.
- b. Acid ingestion
 - (1) The stomach is injured more often than the esophagus.
 - (2) Injury results from coagulation necrosis.
- c. Degree of injury depends on:
 - (1) Type of ingestion (alkali or acid)
 - (2) Volume and concentration of ingested caustic
 - (3) Presence or absence of food in the stomach
 - (4) Tone of the pyloric sphincter

2. Complications

a. Immediate

- (1) Result from caustic injury to the larynx, epiglottis, and occasionally the vocal cords. Immediate airway management may be needed.
- (2) Esophageal perforation is the most frequent complication in the first few days after ingestion. An alkali burn is more likely to cause esophageal perforation than an acid burn.
- (3) Gastric perforation and death can occur within hours of a large acid ingestion; metabolic acidosis, disseminated intravascular coagulation, and hemolysis can also occur with acid ingestion.

b. Delayed

- (1) Esophageal strictures (alkali ingestion)
- (2) Pyloric strictures (acid ingestion)

3. Clinical presentation

- a. Identify and treat life-threatening problems (airway obstruction and hemorrhage). Edema of the larynx, epiglottis, or vocal cords may necessitate cricothyrotomy. However, severe pain is the usual clinical presentation (not respiratory distress or shock).
- b. Assessment of GI injury
 - (1) History of ingestion and any associated symptoms (dysphagia, pain in the mouth, chest, or abdomen)

- (2) Physical examination
 - (a) Check the mouth for oral burns (not sensitive).
 - (b) Check for signs and symptoms of overt or impending esophageal perforation.
 - i. Pleuritic pain located in the chest, epigastrium or back is the single most reliable symptom of an esophageal injury and may be exacerbated by swallowing or neck flexion.
 - ii. Signs of mediastinal air
 - A nasal quality to the voice
 - A systolic, crunching heart sound (Hamman crunch)
 - Subcutaneous emphysema (although classic, may not be present)
4. Diagnostic evaluation: indications for endoscopy
 - a. Any adult patient with a history of alkali or acid ingestion
 - b. Any pediatric patient with:
 - (1) An acid ingestion
 - (2) An alkali ingestion associated with stridor or two or more of the following:
 - (a) Second- or third-degree oral burns
 - (b) Drooling
 - (c) Vomiting
 - (3) Check for evidence of gastric perforation, which can occur within hours of an acid ingestion or massive alkali ingestion
 - (a) Abdominal tenderness, rigidity
 - (b) Decreased bowel sounds
 - (c) Free air on abdominal radiograph (upright or lateral decubitus)
5. Management
 - a. Ensure airway is still patent, and treat hypovolemic shock if present.
 - b. Treat pain.
 - c. Use of diluents
 - (1) The primary indication for the use of diluent (milk or water) is ingestion of solid alkali material (drain openers, automatic dishwashing detergents). Diluting a solid alkali may reduce the degree of tissue injury; in addition, solid alkali material that is adhering to the oropharynx and esophagus needs to be moved to the stomach, where it can be neutralized.
 - (2) Diluents are of no value in the treatment of liquid alkali ingestion, because the tissue injury is immediate.
 - (3) Diluents are of questionable value in the treatment of acid ingestions; although they have been recommended, no studies have demonstrated their benefit.
 - (4) Contraindications
 - (a) Liquid alkali ingestion
 - (b) Vomiting
 - (c) Signs of esophageal or gastric perforation
 - (d) Shock
 - (e) Upper airway obstruction
 - (5) Neutralizers (eg, vinegar, sodium bicarbonate) should never be given, because the resulting exothermic reaction may worsen tissue damage.

- d. The usual postingestion removal measures are contraindicated in the treatment of caustic ingestions.
 - (1) Lavage is contraindicated because of the risk of reexposure of the esophagus and possible perforation of the stomach. In addition, aspiration is a hazard.
 - (2) Charcoal is contraindicated.
 - (a) Alkali and acid are poorly absorbed by charcoal.
 - (b) Charcoal does not prevent tissue injury, because the action of caustics is too rapid.
 - (c) Charcoal interferes with endoscopic visualization of tissue injury.
 - e. Steroid use is controversial but not currently recommended by major toxicology texts.
 - f. Antibiotics should be used only for established infection or for perforation of the upper GI tract.
 - g. Immediate surgical consult should be obtained if GI tract perforation is known/suspected or endoscopy reveals nonviable gastric tissue.
6. Button battery ingestion
- a. If radiography demonstrates lodgement in the esophagus, immediate endoscopic removal is indicated. This is becoming more common with the shift to larger batteries (coin-sized or >20 mm in devices)
 - b. If radiography demonstrates the battery has entered the stomach:
 - (1) The patient can be followed as an outpatient to assure that the battery has passed within 4–7 days.
 - (2) Endoscopic removal is indicated if the battery has not passed through the stomach after 48 hours or at any point that GI symptoms develop.
 - (3) Once the battery passes through the pylorus, the battery can take weeks to pass. The patient should be monitored on an outpatient basis until the battery has passed.

R. Organophosphates

- 1. Pathophysiology
 - a. Organophosphates inhibit acetylcholinesterase, which results in an excess of acetylcholine at the neuronal synapses and myoneural junctions. Excess acetylcholine initially excites—and then paralyzes—neurotransmission at the motor endplate and stimulates CNS muscarinic and nicotinic sites.
 - (1) Muscarinic effects
 - (a) “DUMBBELS”
 - Diarrhea
 - Urination
 - Miosis
 - Bradycardia
 - Bronchorrhea/bronchospasm
 - Emesis
 - Lacrimation
 - Salivation
 - (b) Hypotension and bradycardia

- (2) Nicotinic effects
 - (a) Muscle fasciculations, cramps and weakness, paralysis
 - (b) Hypoventilation and cyanosis
 - (c) Diaphoresis
 - (3) CNS effects
 - (a) Restlessness, respiratory depression
 - (b) Coma, convulsions
 - b. **Organophosphates bind with cholinesterase to form a diethylphosphate bond that can be broken with pralidoxime (2-PAM chloride). If this antidote is not given within 24–36 hours, the cholinesterase molecule may be irrevocably bound ("aging," ie, the antidote function becomes limited), and new cholinesterase will take weeks to regenerate. However, pralidoxime given later may still be beneficial and should be administered.**
- 2. Clinical presentation
 - a. The classic clinical scenario is a farmer with profuse vomiting and diarrhea, pinpoint pupils, and diaphoresis who has a breath odor of insecticide or garlic.
 - b. The "DUMBBELS" syndrome develops first, although patients are usually tachycardic initially and later develop bradycardia. Respiratory insufficiency and sudden respiratory arrest may occur.
 - 3. Diagnostic evaluation
 - a. Should include a plasma or RBC cholinesterase (results will not be available immediately in the emergency department)
 - b. It will be lower than normal in the presence of organophosphate poisoning (<10% of normal value is a serious poisoning).
 - 4. Management
 - a. Supportive care, including airway management
 - b. General detoxification measures
 - (1) Remove clothes, and wash skin thoroughly (medical personnel should protect themselves from skin contamination).
 - (2) Perform lavage if patient has not begun vomiting, and follow with activated charcoal.
 - c. Antidotes
 - (1) Atropine
 - (a) Competitively blocks the muscarinic effects of acetylcholine; it has no effect on the nicotinic receptors at skeletal myoneural junctions and, therefore, will not reverse muscle paralysis.
 - (b) Large doses are frequently needed and should be repeated until tracheobronchial secretions dry up.
 - i. In symptomatic adults, the minimum dose is 2 mg IV every 10–15 minutes as needed; the dose may be doubled every 10 minutes until secretions are controlled.
 - ii. In symptomatic children, the dosage is 0.05 mg/kg IV every 15 minutes as needed. The dose may be doubled every 10 minutes until secretions are controlled.

- (2) Pralidoxime (2-PAM chloride)
 - (a) A biochemical antidote that reactivates the cholinesterase that has been phosphorylated by the organophosphate
 - (b) Patients with signs of cholinergic toxicity should be given pralidoxime as soon as possible even if the precise toxin is not yet known.
 - i. In adults, the initial dose of 1 g IV over 15–30 minutes may be repeated in 1–2 hours as needed.
 - ii. In children, the initial dosage of 25–50 mg/kg IV over 15–30 minutes can be repeated in 1–2 hours.
- (3) Benzodiazepines
 - (a) Seizure prophylaxis (all severely poisoned patients)
 - (b) Also help prevent rhabdomyolysis and discomfort associated with muscle fasciculations
- d. Drugs that should **not** be given to patients with organophosphate toxicity
 - (1) Opioids (worsen respiratory depression)
 - (2) Physostigmine/pyridostigmine (carbamates)
 - (3) Succinylcholine → prolonged paralysis
 - (4) Sympathomimetic agents (to treat bronchospasm, eg, aminophylline)
- 5. Nerve agents
 - a. Originally developed as insecticides prior to World War II; they have been used by the military and, more recently, by terrorists.
 - (1) Cyclohexyl methylphosphonofluoridate (CMPF or GF)
 - (2) Soman (GD)
 - (3) Tabun (GA)
 - (4) Sarin (GB)
 - (5) VX
 - b. Properties that distinguish nerve agents from conventional insecticides
 - (1) High potential for multiple casualties presenting simultaneously, secondary to accidental release from military stockpiles, bioterrorist attacks, or chemical warfare
 - (2) Highly potent (in severe exposures, death can occur within minutes)
 - (3) More rapid “aging” than conventional insecticides, thus limiting the use of pralidoxime as an antidote.
 - c. Management
 - (1) Prehospital
 - (a) Prepare for multiple casualties
 - (b) Activate disaster plan
 - (c) Assemble all appropriate available medications: atropine, glycopyrrolate, pralidoxime, benzodiazepines
 - (2) Hospital decontamination area
 - (a) Protect medical personnel and emergency department from secondary contamination.
 - (b) Ensure surface decontamination of all affected patients before entering emergency department.

- i. Remove clothing.
- ii. Surface washing as indicated.
- iii. Contain contaminated secretions.

(3) Emergency department → pharmacologic therapy (same as above)

S. Mushroom poisoning

1. Distinct clinical classes based on mushrooms that contain:
 - a. Cyclopeptides
 - b. Monomethylhydrazine
 - c. Muscarine
 - d. Coprine
 - e. Psilocybin
2. Diagnostic information
 - a. The most toxic classes have delayed onset of initial symptoms (nausea, vomiting, diarrhea).
 - (1) Cyclopeptides → 6–10 hours after ingestion
 - (2) Monomethylhydrazine → 6–10 hours after ingestion
 - b. Identification of specific mushrooms is very difficult; assistance of the local poison center and a mycologist should be sought.
3. Specific mushroom poisonings
 - a. Cyclopeptide-containing mushrooms
 - (1) Genera: *Amanita phalloides*, *Galerina*, *Lepiota*
 - (2) Clinical effects are predominately renal, hepatic, and CNS.
 - (a) Phase I: nausea, vomiting, and diarrhea, 6–10 hours after ingestion
 - (b) Phase II: symptoms subside but liver function tests are increasing (24–48 hours)
 - (c) Phase III: 1–6 days after ingestion: toxic hepatitis, renal failure, encephalopathy, seizures, coma
 - (3) Management
 - (a) Early recognition, supportive care
 - (b) The following therapies have been used with varying success: multidose activated charcoal, high-dose penicillin G, N-acetylcysteine, milk thistle
 - b. Monomethylhydrazine-containing mushrooms
 - (1) Genus: *Gyromitra esculenta* (false morel)
 - (2) Clinical presentation
 - (a) Nausea and vomiting (6–10 hours after ingestion)
 - (b) Hepatorenal failure
 - (c) Seizures
 - (3) Management
 - (a) General supportive measures and activated charcoal
 - (b) Benzodiazepines and pyridoxine (vitamin B₆)
 - c. Muscarine-containing mushrooms
 - (1) Genera: *Clitocybe* and *Inocybe* (several varieties)
 - (2) Pathophysiology and clinical effects: muscarinic ("DUMBBELS")

- (3) Management
 - (a) Activated charcoal
 - (b) Management geared toward cholinergic toxidrome.
- d. Coprine-containing mushrooms
 - (1) Genus: *Coprinus atramentarius* ("inky cap")
 - (2) Pathophysiology: disulfiram-like effect
 - (3) Clinical presentation: tachycardia, flushing, nausea, and vomiting within 0.5–2 hours of ingestion
 - (4) Management
 - (a) Supportive care
 - (b) Consider activated charcoal
- e. Psilocybin-containing mushrooms
 - (1) Genera: *Psilocybe*, *Gymnopilus*, *Conocybe*, *Panaeolus*
 - (2) Pathophysiology: act on the CNS in a manner similar to that of LSD
 - (3) Clinical presentation: ataxia, hallucinations, hyperkinesia, seizures, nausea and vomiting, mydriasis, tachycardia (within 0.5–2 hours after ingestion)
 - (4) Treatment: benzodiazepines for agitation and seizures

T. Cyanide

- 1. Sources of exposure
 - a. Chemical laboratories → sudden collapse of a worker is suspicious
 - b. Fires
 - (1) Cyanide is a combustion product of wool, silk, polyurethane, synthetic rubber, and nitrocellulose.
 - (2) **Suspect in a fire victim/firefighter/paramedic with decreased level of consciousness, metabolic acidosis, and hypotension.**
 - c. Electroplating
 - d. Precious metal refining
 - e. Photography
 - f. Ingestion of acetonitrile (artificial nail remover) convert to cyanide
 - g. Prolonged exposure to nitroprusside → decreased level of consciousness and metabolic acidosis, or a history of tachyphylaxis to nitroprusside should be suspect.
- 2. Pathophysiology: cyanide interacts with Fe^{+++} in cytochrome aa_3 → blockage of electron transport chain → ↑ ATP production, ↓ H^+ consumption (acidosis), and ↑ lactic acid
- 3. Clinical presentation
 - a. **Neurologic and cardiovascular signs and symptoms predominate the clinical picture.**
 - (1) Neurologic → agitation, seizures, ↓ mental status, ↓ respirations
 - (2) Cardiovascular
 - (a) Bradycardia
 - (b) Hypotension
 - (c) Pulmonary edema
 - b. **Odor of bitter almonds (only 50% of the population can detect this)**
 - c. Gastritis (with ingestions)

- d. **Skin → cherry red color or cyanosis**
 - e. Loss of color difference between retinal veins and arterioles on the fundoscopic exam (arterialization due to high venous pO_2)
4. Management
- a. General measures
 - (1) IV line, oxygen, cardiac monitor
 - (2) Decontamination (unless inhaled)
 - (a) Lavage, activated charcoal
 - (b) Remove clothing and wash skin (hospital personnel should avoid self-contamination; vomitus may give off hydrogen cyanide gas).
 - b. Specific therapy
 - (1) Cyanide antidote kit: contains a 3-step regimen that should be started as soon as cyanide poisoning is suspected.
 - (a) Amyl nitrite pearls: until IV access is obtained, crush into gauze and have patient intermittently inhale.
 - i. Nitrites: administration of nitrates induces methemoglobinemia; cyanide attaches to the methemoglobin.
 - ii. Do not administer amyl nitrite or sodium nitrite to fire victims, because their hemoglobin-oxygen carrying capacity may already be compromised by coexistent carbon monoxide toxicity. Use sodium thiosulfate.
 - (b) 3% sodium nitrite IV at 2.5 mL/min; in anemic patients, dosage may need to be decreased.
 - (c) 25% sodium thiosulfate IV bolus: sodium thiosulfate uses rhodanase (enzyme ubiquitous in body) to convert cyanide and thiosulfate to thiocyanate, which is excreted in the urine.
 - (2) Hydroxocobalamin 5 g IV and repeat once; causes red discoloration of skin and urine; may be used safely in smoke inhalation victims

U. Digitalis

- 1. Pharmacology and mechanism of toxicity
 - a. Cardiac glycosides have a large volume of distribution and a long half-life; elimination is through the kidney; they are not dialyzable.
 - b. Toxicity is via Na^+-K^+ ATPase inhibition → changes in serum electrolytes and cardiac conduction
- 2. Classic clinical scenario
 - a. Weakness, syncope
 - b. Visual changes (yellow halos around lights, referred to as "xanthopsia," is classic)
 - c. Nausea and vomiting (acute toxicity)
 - d. Anorexia (chronic toxicity)
 - e. Hyperkalemia
 - f. ECG changes
 - (1) Bradycardia with AV block (most common finding)
 - (2) Paroxysmal atrial tachycardia with block and bidirectional ventricular tachycardia (most characteristic findings)
 - (3) Although digitalis does not precipitate atrial fibrillation, in patients with chronic poisoning, underlying atrial fibrillation can be regularized.

3. Management

- a. Gastric decontamination (charcoal)
- b. Indications for digoxin Immune Fab
 - (1) Cardiovascular collapse
 - (2) Conduction abnormalities
 - (a) AV block (Mobitz II or third degree)
 - (b) Ventricular dysrhythmias
 - (3) Abnormal chemistries
 - (a) $K^+ > 5.5$ mEq/L (some sources recommend use in patients with $K^+ > 5.0$ mEq/L)
 - (b) Digitalis level > 10 mg/mL
- c. Treatments to avoid
 - (1) Routine supplementation with K^+ or Mg^{++}
 - (2) Transvenous pacing or cardioversion (may induce ventricular dysrhythmias)
 - (3) Standard treatment for hyperkalemia; use of calcium is now thought to be safe in these patients.
 - (4) β -blockers and calcium channel blockers, as well as class IA and class IC drugs (specifically contraindicated; may worsen cardiac conduction)

V. β -Blockers

- 1. Produce toxicity through β_1 - or β_2 -receptor blockade
- 2. Classic clinical scenario
 - a. Coma/seizures
 - b. Hypotension/bradycardia
 - c. Hypoglycemia
 - d. ECG changes reflect sodium- or potassium-channel blockade
 - (1) Widened QRS
 - (2) Prolonged QT interval
- 3. Management
 - a. Gastric decontamination
 - b. Specific therapy for cardiovascular collapse
 - (1) Vasopressors
 - (2) Atropine can be given but may be ineffective.
 - (3) Glucagon 2–5 mg IV bolus, then a 2–5 mg/hr infusion
 - (4) Calcium chloride 20 mg/kg bolus, then a 20 mg/kg/hr infusion
 - (5) High-dose insulin therapy (see calcium channel blockers, below)
 - (6) IV lipid emulsion (see calcium channel blockers, below)
 - (7) Pacing at a modest rate to allow channels to recycle
 - (8) Intra-aortic balloon pump
 - c. Contraindicated drugs
 - (1) Calcium channel blockers
 - (2) Class IA and class IC agents

W. Calcium channel blockers

1. Inhibit calcium channel conduction in both smooth and cardiac muscle, including the cardiac conduction system.
2. Classic clinical scenario
 - a. Hypotension and bradycardia, especially with diltiazem, verapamil; may have reflex tachycardia with the dihydropyridines
 - b. Hyperglycemia
 - c. Metabolic acidosis
 - d. Pulmonary edema
 - e. Ileus
 - f. ECG changes reflect conduction delay (AV dissociation is common)
 - (1) Prolonged PR interval
 - (2) Bradycardia, bradydysrhythmias
3. Management
 - a. Decontamination
 - (1) Charcoal may be considered.
 - (2) Whole-bowel irrigation (if sustained-release preparation)
 - b. Specific therapies
 - (1) Epinephrine (best vasopressor)
 - (2) Atropine (may be ineffective)
 - (3) Glucagon 0.05–0.15 mg/kg bolus, then a 2–5 mg/hr infusion
 - (4) Calcium
 - (5) Pacing at a modest rate to allow channels to recycle
 - (6) Intra-aortic balloon pump
 - c. High-dose insulin (0.2–2 units/kg/hr) should be started in any patient not responding to IV fluids and calcium administration.
 - d. Intravenous lipid emulsion is becoming more popular as evidence-based medicine demonstrates increased efficacy for a variety of drug toxicities. Dosage (20% solution): 1.5 mL/kg bolus, followed by 0.25 mL/kg/min for 30–60 minutes.
 - e. Because of the prolonged absorption time of sustained-release preparations, 24-hour observation is mandatory.

X. Carbon monoxide

1. Primary mechanism of toxicity is carbon monoxide binding to cytochrome oxidase, disrupting oxidative phosphorylation, and interference with microvascular homeostasis. Of secondary importance is carbon monoxide binding to hemoglobin, which subsequently decreases the oxygen-carrying capacity of the blood.
2. Clinical presentation
 - a. Mild to moderate poisoning
 - (1) Weakness
 - (2) Headache, dizziness
 - (3) Nausea, vomiting
 - b. Severe poisoning
 - (1) Syncope

- (2) Visual complaints
- (3) Chest pain
- (4) Dysrhythmias
- (5) Coma, seizures
- (6) Cardiovascular collapse
- c. Flu-like symptoms are common, accounting for frequent misdiagnosis. Diagnostic clues:
 - (1) Flu without fever
 - (2) Multiple victims from same dwelling
 - (3) Small household pets ill or dead from unknown cause
 - (4) Recent vehicle travel
 - (5) Early winter when furnaces are being turned on
 - (6) Power outages in winter
- d. Delayed neuropsychiatric sequelae are seen in 10%–43% of cases after apparent recovery
 - (1) Headache, dizziness
 - (2) Memory deficits
 - (3) Personality alterations
 - (4) Parkinsonism
- 3. Diagnostic evaluation
 - a. Serum carboxyhemoglobin levels
 - (1) May not correlate well with toxicity, especially if drawn after empiric oxygen administration
 - (2) Venous levels may be used.
 - b. Carbon monoxide neuropsychiatric screening battery is a sensitive indicator of toxicity in adults.
 - c. CT or MRI may show bilateral hypodensity in the basal ganglia, diffuse white matter hypodensities, or cerebral edema.
- 4. Management: oxygen therapy
 - a. 100% oxygen by tight-fitting mask for 4 hours is indicated when the carboxyhemoglobin level is <20%; longer therapy is indicated for infants and in pregnant patients.
 - b. Hyperbaric oxygen should be considered if any of the following conditions are present:
 - (1) Carboxyhemoglobin level >25%
 - (2) Carboxyhemoglobin level >10% if pregnant
 - (3) Any neurologic symptom other than mild headache, including brief loss of consciousness, focal neurologic deficit, seizure
 - (4) Coma
 - (5) Myocardial ischemia
 - (6) Worsening symptoms despite oxygen therapy
 - c. Half-life of carbon monoxide during treatment with various oxygen sources
 - (1) Room air: 240 minutes
 - (2) Nonrebreather: 90 minutes
 - (3) Hyperbaric oxygen: 20 minutes

Y. Mercury

1. Forms

- a. Elemental (liquid mercury, quicksilver → thermometers, barometers, sphygmomanometers)
- b. Inorganic (mercuric chloride) → chemistry laboratories
- c. Organic (methylmercury) → fish and shellfish

2. Pathophysiology

a. Elemental (CNS and lung)

- (1) Acute inhalation → pulmonary edema
- (2) Chronic exposure → tremor, acrodynia, neuropsychiatric changes
- (3) Contamination
 - (a) Elemental mercury is most toxic when inhaled (especially if volatilized by heat).
 - (b) Ingestions (eg, broken thermometer in mouth) are nontoxic unless extravasation into the peritoneum occurs.

b. Inorganic (skin, GI tract, and kidney)

- (1) Corrosive to skin
- (2) Highly toxic when ingested
 - (a) Hemorrhagic gastroenteritis → shock
 - (b) Acute tubular necrosis

c. Organic (chronic placental-fetal transfer → CNS): profound neurotoxicity to the developing brain

3. Management

- a. Decontamination for ingestion of inorganic and organic mercury: despite the corrosiveness of inorganic mercury (and potential risk of perforation), the benefit of GI decontamination outweighs the risk.
 - (1) If mercury has been ingested within the last 2 hours, lavage with a small orogastric (or nasogastric) tube.
 - (2) When mercury is seen on plain radiograph of the abdomen, whole-bowel irrigation is indicated.
- b. Chelation for suspected ingestion (do not wait for blood levels)
 - (1) For all forms of mercury ingestion, may use dimercaptosuccinic acid (DMSA), administered orally
 - (2) For significant, acute toxicity and/or inability to tolerate oral therapy, use BAL (dimercaprol) until the patient can tolerate DMSA.

Z. Sulfonylureas

1. Examples (all are oral agents): glipizide, glimepiride, glyburide, chlorpropamide
2. Pathophysiology: primary mechanism is binding to pancreatic β cells and stimulating endogenous insulin secretion
3. Clinical presentation
 - a. Signs and symptoms of hypoglycemia (eg, tachycardia, tremor, diaphoresis, confusion, agitation, seizures, coma)
 - b. Can range from mild to severe and may be delayed
 - c. May not manifest in children, the elderly, those taking β -adrenergic receptor blockers; must check blood glucose levels!

4. Diagnostic evaluation
 - a. Rapid bedside glucose determination
 - (1) Can be unreliable at extreme hypo- and hyperglycemia
 - (2) Check frequently (less often as clinical scenario evolves and stabilizes)
 - b. Electrolytes: potassium and magnesium levels can also be depressed.
5. Management
 - a. Activated charcoal in the appropriate clinical conditions.
 - b. Supplemental glucose
 - (1) Should be administered *only* to hypoglycemic patients. In healthy patients with sulfonylurea poisoning, administration of prophylactic glucose can precipitate severe hypoglycemia, because these drugs may facilitate exaggerated insulin secretion in response to a glucose load.
 - (2) Repeated boluses and/or initiation of a glucose infusion (with D5 or D10) may be necessary
 - c. Octreotide
 - (1) Synthetic, long-acting analog of somatostatin that antagonizes pancreatic insulin secretion
 - (2) Monoamine oxidase
 - (3) Indications: hypoglycemic sulfonylurea-poisoned patients whose serum glucose concentrations cannot be maintained with a dextrose infusion. Some recommend its use as a first-line agent along with glucose administration.
 - (4) Available for administration by IV or SC routes only
 - d. Alkalinization of the urine has been used to enhance renal elimination of chlorpropamide but is of no value for treatment of poisoning by other sulfonylureas.

TOXICOLOGIC DISORDERS: PRACTICE CLINICAL SCENARIOS

Answers immediately follow the practice clinical scenarios.

Scenario A

Presentation: A 36-year-old woman presents via EMS for evaluation of vomiting and lethargy. The paramedics report that the woman's family has been ill with flu-like symptoms. The patient has had no fever, diarrhea, or cough, and no significant past medical history.

Physical examination: Vital signs are temperature 98.2°F (36.8°C), heart rate 85 beats per minute, respiratory rate 18 breaths per minute, and blood pressure 122/60 mmHg. The patient appears sleepy and pale. Pupils are equal and reactive to light. Chest auscultation demonstrates clear lung fields and regular cardiac rate and rhythm. On neurologic examination, the patient is oriented to self and place but has some difficulty providing some details of the history. She has no focal deficits. An initial blood glucose level is 90 mg/dL.

What is the diagnosis?

Scenario B

Presentation: A 2-year-old boy presents with his parents for evaluation of abdominal pain, vomiting, and copious bloody diarrhea. The father reports that the boy was found playing under the bathroom sink, where his mother's prenatal vitamins are stored.

Physical examination: Vital signs are temperature 100°F (38°C), heart rate 144 beats per minute, respiratory rate 30 breaths per minute, and blood pressure 60/palp. The child weighs 44 pounds (20 kg). He is pale, diaphoretic, and barely responsive to stimuli. Chest auscultation demonstrates clear lung fields and regular cardiac rate and rhythm with no murmur. Capillary refill time is 5 seconds. Examination of the abdomen reveals mild, diffuse tenderness without peritoneal signs. Rectal examination is positive for gross blood. The child is somnolent, with no focal deficits.

Diagnostic evaluation

Arterial blood gases: 7.12 | 90 | 22 | 9

WBC = 18,300/mm³, hemoglobin = 8.9 mg/dL, hematocrit = 26

Na⁺ = 135 mEq/L, Cl⁻ = 98 mEq/L, K⁺ = 4.5 mEq/L, HCO₃⁻ = 8

Glucose: 54 mg/dL

BUN: 38 mg/dL

What is the diagnosis?

Scenario C

Presentation: A 22-year-old woman presents after ingesting over 100 tablets of acetaminophen in a suicide attempt 4 hours before arrival. She denies coingestants and complains of nausea. She has no past medical history.

Physical examination: Vital signs are temperature 98.7°F (37°C), heart rate 82 beats per minute, respiratory rate 14 breaths per minute, and blood pressure 123/71 mmHg. The patient is tearful but in no acute distress. Pupils are 3 mm and reactive to light, and mucus membranes are moist. Chest auscultation demonstrates clear lung fields and regular cardiac rate and rhythm with no murmurs. Bowel sounds are normal, and the abdomen is nontender on palpation. Neurologic examination is unremarkable.

What is the diagnosis?

Scenario D

Presentation: A 20-year-old man presents with a friend 1 hour after drinking a quart of windshield wiper solution in a suicide attempt. The patient denies coingestants, vision changes, abdominal pain, nausea, and vomiting. He has no past medical history.

Physical examination: Vital signs are temperature 97.8°F (37°C), heart rate 79 beats per minute, respiratory rate 32 breaths per minute, and blood pressure 124/77 mmHg. The patient is intoxicated but in no distress. Pupils are equal and reactive to light. Chest auscultation demonstrates clear lung fields and regular cardiac rate and rhythm. Bowel sounds are normal, and the abdomen is nontender on palpation. On neurologic examination, visual acuity is 20/20 bilaterally, the patient is oriented to person and place, cranial nerves II–XII are intact, and no focal deficits are seen.

What is the diagnosis?

Scenario E

Presentation: An 82-year-old man presents for evaluation of "not feeling right." His wife states that he has become increasingly confused over the last 3 days. He has also been very irritable, unsteady on his feet, and seems to be breathing very rapidly and deeply at times. They deny a history of recent or past trauma. Past medical history includes hypertension, hyperlipidemia, and coronary artery disease, and medications include hydrochlorothiazide, simvastatin, aspirin, diltiazem, and nitroglycerin as needed.

Physical examination: Vital signs are temperature 100.2°F (38°C), heart rate 99 beats per minute, respiratory rate 32 breaths per minute, and blood pressure 154/88 mmHg. The patient is alert, with no signs of trauma. His face is flushed, and mucous membranes are dry. His speech is slurred, but there is no facial drop. Pupils are equal and reactive to light. Chest auscultation demonstrates clear lung fields and regular cardiac rate and rhythm. Bowel sounds are normal, and the abdomen is nontender on palpation. On neurologic examination, motor strength is symmetric in upper and lower extremities. There is no clonus, and Babinski responses are down going bilaterally. Cranial nerves are intact. The patient appears to have ataxia with transfer into hospital bed; however, gait testing was not attempted because of his unsteadiness. Cardiac monitor shows normal sinus rhythm.

What chronic poisoning can manifest in this fashion?

ANSWERS TO PRACTICE CLINICAL SCENARIOS

Scenario A

Diagnosis: carbon monoxide poisoning

Diagnostic evaluation: A carboxyhemoglobin level is essential to the diagnosis of carbon monoxide poisoning. Arterial or venous blood samples may be used.

Management: The patient should be given high-flow oxygen, and hyperbaric oxygen should be considered. Hyperbaric oxygen therapy decreases the rate of delayed neurologic sequelae in carbon monoxide-poisoned patients and reduces the half-life of carbon monoxide to 20 minutes, versus a half-life of 240 minutes when a patient is breathing room air.

Scenario B

Diagnosis: iron poisoning

Diagnostic evaluation: Iron poisoning is classically described as occurring in five sequential "stages."

- First stage: first few hours after ingestion; direct irritative effect of iron on GI tract causes GI upset, including vomiting and diarrhea (possibly bloody). The absence of these symptoms within 6 hours of ingestion essentially excludes the diagnosis.
- Second stage: may last for up to 24 hours; GI symptoms improve, but patient will not become asymptomatic and may have abnormal vital signs and evidence of poor perfusion.
- Third stage: may appear early or develop hours after the second stage; shock and metabolic acidosis develop, bleeding and hypovolemia may worsen, and hepatic dysfunction, cardiomyopathy, and renal failure may occur.
- Fourth stage: 2–5 days after ingestion; transaminases increase and may progress to hepatic failure.
- Fifth stage: 4–6 weeks after ingestion; involves gastric outlet obstruction secondary to corrosive effects of iron on pyloric mucosa.

Management: A serum iron concentration should be obtained. Serum iron concentrations of 300–and 500 mcg/dL correlate with mild symptoms, 500–1,000 mcg/dL with moderate symptoms, and >1000 mcg/dL with severe symptoms. GI decontamination with orogastric lavage can be considered. Whole-bowel irrigation can be performed in standard fashion. Iron does not adsorb to activated charcoal. Deferoxamine (IV infusion) is the chelator of choice for severe iron poisoning and can be repeated every 4–6 hours as needed. Deferoxamine removes iron from tissues and free iron from plasma and is safe in children and in pregnancy. Give empirically for critically ill patients.

Scenario C

Diagnosis: acetaminophen toxicity

Diagnostic evaluation: An acetaminophen level should be obtained and plotted on the Rumack-Matthew nomogram (can be used only after a single acute ingestion). A 4-hour level >150 mcg/mL is considered toxic; the nomogram will allow prediction of potentially toxic acetaminophen levels between 4 and 24 hours after acute ingestion.

Management: GI decontamination with activated charcoal is recommended by some in the appropriate clinical conditions (patient protecting airway). Many forgo administration of activated charcoal given the high efficacy of N-acetylcysteine (NAC). Its administration may be more important when the presence of coingestants is of concern. Administration of activated charcoal is not thought to significantly hinder the efficacy of oral NAC, and no dosing adjustments to either need be made. If the patient's acetaminophen level is above the treatment line on the Rumack-Matthew nomogram, NAC should ideally be administered. NAC is most effective when initiated within 8 hours of ingestion but is still very effective at reducing toxicity in later presenting patients and should not be withheld.

NAC is available in both oral and IV forms and is dosed as follows:

- Oral: 140 mg/kg, then 70 mg/kg every 4 hours for 17 doses (total 72 hours)
- IV: 150 mg/kg, then 50 mg/kg over 4 hours, then 100 mg/kg over 16 hours (total 21 hours)

Scenario D

Diagnosis: methanol toxicity

Diagnostic evaluation: Common presentations include suicidal ideation, an ethanol-dependent patient who has limited access to ethanol, or a pediatric patient with accidental ingestion of windshield wiper fluid and/or antifreeze. Symptoms may present 12–18 hours after ingestion or longer if ethanol is also consumed and may include visual disturbances (looking at a snowstorm) and abdominal discomfort.

Management: Management includes obtaining a methanol level (and an ethylene glycol level if the history does not confirm that methanol was the ingested). Fomepizole should be given for proven or suspected levels >20 mg/dL or in patients requiring dialysis. Hemodialysis is indicated for signs of significant toxicity, such as the presence of anion gap metabolic acidosis, methanol level >50 mg/dL, or signs of ocular toxicity.

Scenario E

Diagnosis: aspirin toxicity

Diagnostic evaluation: Other common presentations include acute aspirin ingestion as a suicide attempt, chronic ingestion for pain control, and/or pediatric exploratory ingestion of substances containing salicylate (oil of wintergreen). Consider aspirin poisoning in any patient with unexplained nonfocal neurologic and behavioral abnormalities, especially with coexisting acid-base disturbance, tachypnea, dyspnea, or noncardiogenic pulmonary edema. Chronic poisoning is usually seen in the elderly and may cause progressive neurologic dysfunction and cerebral edema, renal failure, and pulmonary edema/ARDS.

Management: A serum salicylate level should be obtained. Other clues to the diagnosis include presence of a mixed acid-base disorder. Salicylates cause hyperventilation because of action on the medullary respiratory center (early finding) with resultant primary respiratory alkalosis. Later, salicylates cause uncoupling of oxidative phosphorylation, resulting in a shift toward anaerobic metabolism and ultimately leading to metabolic acidosis (excess lactate production) and heat production.

In severe ingestion, hourly monitoring of salicylate, urine pH, potassium levels, and blood pH (arterial or venous) is indicated. Gastric decontamination should be considered, including orogastric lavage and activated charcoal. Alkalinization of serum and urine should be done to enhance salicylate elimination and prevent salicylate from entering CNS: 1–2 mEq/kg of sodium bicarbonate (bolus) followed by 150 mEq in 1 L of D5W infused at 1.5–2 times maintenance rate. Adjust to achieve urine pH ≥ 7.5 . Hemodialysis is indicated for:

- Salicylate levels >80 – 100 mg/dL (acute poisoning) or >40 – 60 mg/dL (chronic poisoning)
- Worsening clinical condition (severe acidosis, hypotension, shock) despite appropriate, aggressive treatment with sodium bicarbonate
- Evidence of end organ injury
 - CHF/fluid overload that prevents patient from tolerating fluids necessary for urinary alkalinization
 - Renal failure, which may prevent patient from tolerating fluid load and may prevent patient from eliminating a sufficient amount of salicylate
 - Seizures
 - Consider for patients who require endotracheal intubation

ENDOCRINE, METABOLIC, AND NUTRITIONAL DISORDERS

ADULT FLUID VOLUMES	755
Normal Values.....	755
Patient Assessment	755
FLUID AND ELECTROLYTE DISORDERS	756
Basic Science Review	756
Total Body Water/Fluid Compartments.....	756
Water Regulation.....	756
Sodium Regulation	758
Potassium Regulation.....	760
Calcium Regulation	760
Magnesium Regulation	761
Chloride Regulation.....	761
Physiologic "Pumps" that Maintain Electrolyte Balance	761
The Anion Gap	761
Pathophysiology of Acid-Base Disorders	762
Electrolyte Disorders.....	764
Hyponatremia	764
Hypernatremia	767
Dehydration	768
Hypokalemia.....	769
Hyperkalemia.....	770
Hypocalcemia	772
Hypercalcemia	773
Hypomagnesemia.....	774
Hypermagnesemia.....	776
Hypochloremia.....	776
Hyperchloremia.....	777
Acid-Base Disorders.....	777
General Approach to the Patient with an Acid-Base Abnormality	777
Metabolic Acidosis	779
Metabolic Alkalosis	781
Respiratory Acidosis	781
Respiratory Alkalosis.....	782
Lactic Acidosis.....	782

Important Points to Remember in the Management of Acid-Base Disorders	783
Clinical Situations and Associated Laboratory Findings	784
METABOLIC DISORDERS	785
Hypoglycemia	785
Diabetic Ketoacidosis.....	787
Hyperglycemic Hyperosmolar Nonketotic Coma.....	791
Alcoholic Ketoacidosis	793
Thyroid Storm	794
Myxedema (Hypothyroid) Coma.....	796
Adrenal Insufficiency (Addison Disease) and Crisis.....	799

ENDOCRINE, METABOLIC, AND NUTRITIONAL DISORDERS: SELF-ASSESSMENT QUESTIONS

1. The major intracellular cation is:
 - (a) Potassium
 - (b) Calcium
 - (c) Chloride
 - (d) Sodium
2. The weight of total body water in a 70-kg adult man is closest to:
 - (a) 40 kg
 - (b) 20 kg
 - (c) 60 kg
 - (d) 30 kg
3. All of the following are associated with an increased difference between the calculated and measured serum osmolality (osmolal gap) except:
 - (a) An ethanol level of 200mg%
 - (b) Administration of 100 grams of mannitol
 - (c) Hyperglycemic nonketotic coma
 - (d) Severe ketoacidosis
4. All of the following are true about antidiuretic hormone (ADH) except:
 - (a) It is released in response to decreases in serum osmolality.
 - (b) It is released in response to decreases in intravascular volume.
 - (c) It acts on the renal tubules to decrease free water excretion.
 - (d) It is present in excessive amounts in syndrome of inappropriate ADH secretion (SIADH).
5. All of the following may result in changes in the concentration of ionized calcium except:
 - (a) Decreased serum albumin
 - (b) Hyperventilation
 - (c) Excessive parathyroid hormone
 - (d) Vitamin D intoxication
6. The regulation and function of magnesium are closely tied to:
 - (a) Sodium
 - (b) Chloride
 - (c) Calcium and phosphate
 - (d) Serum albumin

7. Causes of an anion gap acidosis include all of the following except:
- (a) Salicylate poisoning
 - (b) Isopropyl alcohol ingestion
 - (c) Uremia
 - (d) Methanol poisoning
8. An increased anion gap (>16 mEq/dL) and an increased osmolal gap (>10 mOsm/dL) may be seen in all of the following except:
- (a) Uremia
 - (b) Ethanol intoxication
 - (c) Methanol poisoning
 - (d) Diabetic ketoacidosis
9. The pulmonary excretion of CO_2 (hyperventilation):
- (a) Increases the serum H^+ concentration
 - (b) Increases the serum pH
 - (c) Decreases the renal excretion of bicarbonate
 - (d) Increases the serum concentration of bicarbonate
10. Physiologic compensation for metabolic acidosis occurs through all of the following mechanisms except:
- (a) Persistent vomiting
 - (b) Pulmonary excretion of CO_2
 - (c) Increased renal H^+ excretion
 - (d) Increased renal bicarbonate losses
11. In human studies and experimental animal models, central pontine myelinolysis has been associated with all of the following except:
- (a) Rapid correction of symptomatic hyponatremia (<24 hours duration)
 - (b) Correction of hyponatremia >2 days duration at a rate >0.6 mEq/L/hour
 - (c) Correction of hyponatremia >2 days duration at a rate >25 mEq over 48 hours
 - (d) Correction of hyponatremia >24 hours duration at a rate >2.5 mEq/hour
12. An 80-year-old woman is found unconscious in her Houston apartment, which is not air-conditioned, in August. Her serum sodium is 185 mEq/L. Blood pressure is 60 mmHg by palpation, and pulse 130 beats per minute. The most appropriate fluid regimen for her initial resuscitation is:
- (a) D5W at 500 mL/hour
 - (b) D5/0.45% normal saline at 250 mL/hr
 - (c) Normal saline or lactated Ringer's 1 L in the first hour
 - (d) D5/0.33% normal saline at 500 mL/hr

13. A 60-year-old man with a history of congestive heart failure presents to the emergency department complaining of pedal edema. His mental status is clear. Blood pressure is 120/80 mmHg, and pulse is 80 beats per minute. Lungs are clear, and neck veins distended. Serum sodium is 105 mEq/L. Of the following therapies, which is the most appropriate?
- (a) Infusion of 3% normal saline at 50 mL/hour and concomitant administration of furosemide
 - (b) Infusion of normal saline at 200 mL/hour with concomitant administration of furosemide
 - (c) Water restriction
 - (d) IV administration of 40 mg of furosemide every hour until serum sodium is normal
14. A 2-year-old child has been vomiting for 6 days. In the emergency department she is listless, with a pulse of 180 beats per minute and capillary refill >4 seconds. Her weight is 10 kg. The serum sodium is 120 mEq/L. The most appropriate initial therapy is:
- (a) Infusion of 3% normal saline at 1 mL/kg/hour and concomitant administration of furosemide
 - (b) Infusion of 3% normal saline at 1 mL/kg/hr
 - (c) A rapid infusion of normal saline at 20 mL/kg
 - (d) D5/0.45% normal saline at 125 mL/hr
15. Of the following clinical scenarios, which patient has the most urgent need for rapid potassium replacement?
- (a) A 4-year-old with persistent vomiting, metabolic alkalosis, and a serum potassium of 3.0 mEq/L
 - (b) A 56-year-old woman taking a diuretic and digoxin, with a serum potassium of 3.0 mEq/L
 - (c) A 22-year-old insulin-dependent diabetic patient with an arterial pH of 6.9 and a serum potassium of 3.0 mEq/L
 - (d) A 45-year-old man with delirium tremens and a serum potassium of 3.0 mEq/L
16. A 60-year-old man presents with confusion, polyuria, and a serum calcium of 16 mg/dL. Of the following therapies, which is the least appropriate?
- (a) Normal saline IV at 500 mL/hour
 - (b) Salmon calcitonin 4 U/kg IM
 - (c) Methylprednisolone sodium succinate 100 mg IV
 - (d) Vitamin D 25,000 units IM
17. A healthy 17-year-old woman suffers a cardiac arrest during an infusion of magnesium sulfate for treatment of eclampsia. In addition to starting CPR, the most appropriate initial treatment is:
- (a) Immediate hemodialysis
 - (b) IV infusion of 1 g calcium chloride
 - (c) IV infusion of 140 mEq of sodium bicarbonate
 - (d) IV infusion of 10 mEq of potassium chloride

18. A 50-year-old alcoholic patient presents with obtundation. Blood pressure is 150/100 mmHg, and pulse is 120 beats per minute. He is treated empirically with 25 g of D50W and 100 mg of thiamine. The pH is 7.2, $p\text{CO}_2 = 15$, $\text{Na}^+ = 140 \text{ mEq/L}$, $\text{Cl}^- = 102 \text{ mEq/L}$, $\text{HCO}_3^- = 6 \text{ mEq/L}$, $\text{K}^+ = 4 \text{ mEq/L}$, and glucose = 250 mg/dL. Urine ketones are negative. Ethanol is not detectable. The osmolal gap is 100 mOsm/L. The most urgent interventions are:
- (a) Administration of 1 mEq/kg of sodium bicarbonate, blood cultures, and broad-spectrum antibiotics, followed by observation in the ICU
 - (b) Initiation of an ethanol infusion (or administration of 4-methylpyrazole) and arrangements for hemodialysis
 - (c) Immediate head scan and neurosurgical consultation
 - (d) Intubation and gastric lavage, followed by administration of 60 g of activated charcoal with sorbitol
19. The mental obtundation seen in association with nonketotic hyperosmolar coma is most closely associated with:
- (a) Serum bicarbonate level
 - (b) Serum osmolality
 - (c) pH of the plasma
 - (d) pH of the cerebrospinal fluid
20. The nitroprusside test for ketones most accurately assesses the presence of:
- (a) Acetone
 - (b) Acetoacetate
 - (c) β -hydroxybutyrate
 - (d) It assesses all of the above equally well.
21. Which of the following drugs is not classically associated with producing drug-induced hypoglycemia?
- (a) Alcohol
 - (b) Aspirin
 - (c) NSAIDs
 - (d) Disopyramide
22. Expected findings in a patient with purely alcoholic ketoacidosis include all of the following except:
- (a) High anion gap acidosis
 - (b) Markedly increased ($>300 \text{ mg/dL}$) serum glucose
 - (c) A nitroprusside test that may be only weakly positive
 - (d) Nondetectable or low blood alcohol level

23. The mainstay of therapy for patients in alcoholic ketoacidosis is IV hydration with:
- (a) A saline solution (normal saline or 0.45% normal saline)
 - (b) A glucose solution (D5W)
 - (c) A solution containing both glucose and saline (D5/normal saline or D5/0.45% normal saline)
 - (d) The type of solution used is irrelevant.
24. The acidosis seen in association with alcoholic ketoacidosis is primarily attributable to the presence of:
- (a) β -hydroxybutyrate
 - (b) Acetoacetate
 - (c) Acetone
 - (d) Lactate
25. All of the following ingestions are associated with a high anion gap acidosis except:
- (a) Ethylene glycol
 - (b) Paraldehyde
 - (c) Aspirin
 - (d) Isopropyl alcohol
26. A patient presents with hypertension, tachycardia, a fever, and slight confusion. You suspect thyroid pathology. Which of the following drugs should be avoided?
- (a) Aspirin
 - (b) Propranolol
 - (c) Ibuprofen
 - (d) Acetaminophen
27. The role of propylthiouracil in the treatment of thyroid storm is most accurately described as that of:
- (a) Inhibiting conversion of T_3 to T_4
 - (b) Retarding release of stored thyroid hormone
 - (c) Blocking the synthesis of thyroid hormone
 - (d) None of the above
28. All of the following statements regarding hypothyroidism are accurate except:
- (a) Primary hypothyroidism is more common than secondary hypothyroidism.
 - (b) Patients with secondary hypothyroidism have increased levels of thyroid-stimulating hormone.
 - (c) Treatment of Grave disease is the most common cause of primary hypothyroidism in adults.
 - (d) Drugs that have been associated with development of primary hypothyroidism include lithium and phenylbutazone.

29. After abrupt cessation of steroid therapy, a patient presents with vomiting, hypotension, and generalized malaise. All of the following laboratory findings would be suspected except:

- (a) Hyponatremia
- (b) Hypoglycemia
- (c) Hyperkalemia
- (d) Hypocalcemia

ANSWERS

1. a	7. b	13. c	19. b	25. d
2. a	8. b	14. c	20. b	26. a
3. c	9. b	15. c	21. c	27. c
4. a	10. d	16. d	22. b	28. b
5. a	11. a	17. b	23. c	29. d
6. c	12. c	18. b	24. a	

Use the pre-chapter multiple choice question worksheet (page xx) to record and determine the percentage of correct answers for this chapter.

ADULT FLUID VOLUMES

I. NORMAL VALUES

- A. Extracellular fluid = 200 mL/kg
- B. Estimated blood volume = 70 mL/kg
- C. Plasma volume = 35.5 mL/kg (approximately half the blood volume)

II. PATIENT ASSESSMENT

- A. Normal capillary refill (nailbeds) ≤ 2 seconds
 - 1. If delayed >2 seconds, a volume deficit $\geq 15\%$ is present.
 - 2. **Capillary refill can also be delayed from poor pump function.**
- B. Acute onset tachycardia
 - 1. >100 beats per minute = 15%–30% volume deficit
 - 2. >120 beats per minute = 30%–40% volume deficit
 - 3. >140 beats per minute = $>40\%$ volume deficit
- C. Orthostatic pulse and blood pressure changes = at least a 20% volume deficit
 - 1. Orthostatic vital signs are poorly sensitive and specific.
 - 2. **Systemic orthostasis is a more reliable finding.**
- D. Hypotension = at least a 30% volume deficit. **Pulse pressure (gap between systolic and diastolic) usually narrows before systolic blood pressure drops.**
- E. Pulmonary artery wedge pressure monitoring is the most accurate method of determining fluid volume status, especially in patients with pulmonary disease, right ventricular failure, and cardiac tamponade, **but Swan-Ganz catheters are rarely used today.**
 - 1. Central venous pressure monitoring is an acceptable alternative in patients without these disorders.
 - 2. The use of central venous pressure monitoring in shock states continues to be a controversial issue.

FLUID AND ELECTROLYTE DISORDERS

I. BASIC SCIENCE REVIEW

A. Total body water/fluid compartments

1. Total body water = 60% total body weight (adult man)
2. Proteins are nondiffusible; carrying a large negative charge, they hold a large number of cations inside the cell.
3. Intracellular water = $\frac{2}{3}$ total body water (40% of body weight)
4. Energy-dependent (ATP) Na^+ and K^+ are the primary effectors of electrolyte gradients between intracellular and interstitial spaces.
5. Extracellular water = $\frac{1}{3}$ total body water (20% of body weight)

Table 31: Total Body Fluids

Intracellular fluid	(mEq/L)	
K^+	150	
Proteins	75	
Mg^{++}	30	
Na^+	14	75%
HCO_3^-	10	
Cl^-	4	
Ca^{++}	<1	
Interstitial fluid	(mEq/L)	
Na^+	140	
Cl^-	113	
HCO_3^-	27	
K^+	5	
Mg^{++}	3	
Ca^{++}	9	
Plasma		25%

B. Water regulation

1. Definitions

a. **Solvent:** in physiologic systems, this is H_2O .

b. **Solute:** crystalloid (cation, anion) or colloid (plasma protein)

c. **Osmosis:** the movement of solvent (H_2O) across membranes along an osmolar gradient

d. **Osmolality:** a reflection of the total number of particles in solution

2. Physiologic concepts

- a. In a water-based system, 1 L = 1 kg.
- b. Semipermeable membranes allow the passage of solvent (H_2O) but not solute (electrolytes, plasma protein), thus permitting free movement of water between the interstitial/plasma compartments and the intracellular/extracellular compartments.
- c. At equilibrium, the concentration of molecules in the fluid of one compartment is equal to that in all the other compartments. When free water is lost from one compartment, its concentration of molecules increases; this causes water to diffuse into that compartment until the concentrations equalize.
- d. Normal serum osmolality is 275–295 mOsm/L.
 - (1) Sodium, chloride and bicarbonate are the major contributors, although glucose and BUN must be taken into account. Because $[Na^+]$ approximates $[Cl^- + HCO_3^-]$, the following equation may be used to calculate serum osmolality:

$$(2 \times Na) + \frac{BUN}{2.8} + \frac{Glucose}{18} + \frac{Ethanol}{4.6}$$

The calculated value should be within 10 mOsm/L of the measured value. The difference between the measured and calculated values is the osmolal gap. A high osmolal gap increases suspicion of an unidentified substance dissolved in the blood.

(2) Causes of abnormal osmolar states:

(a) Hyperosmolality = \uparrow measured serum osmolality

- i. \uparrow serum Na^+ (no gap)
- ii. Alcohol ingestion: either ethanol or "toxic alcohols" (large gap)
 - Most common cause of coma + \uparrow serum osmolality
 - \uparrow serum osmolality + \uparrow osmolal gap + anion gap acidosis \uparrow methanol or ethylene glycol
 - Isopropyl alcohol causes an osmolal gap but no acidosis.
- iii. Hyperosmolar hyperglycemic nonketotic coma (no gap)
- iv. Ketoacidosis (small gap)
- v. Uremia (small gap)

(b) Hyposmolality $\rightarrow \downarrow$ serum Na^+ = \downarrow measured serum osmolality

3. Clinical pathophysiology

a. The average adult requires about 2,000–3,000 mL of water per day (depending on losses).

b. Water loss

(1) The volume of water loss per day can be divided into two categories:

(a) Insensible losses

- i. Respiratory tract (~600 mL)
- ii. Skin (~300 mL)
- iii. Feces (~100 mL)

(b) Urinary losses (~1,500 mL)

- i. Normal dietary intake produces ~300 mL of solute/day.
- ii. Maximal concentrating ability of the normal nephron is 1,200 mOsm/L. To maintain normal electrolyte balance and excrete waste, the average adult

must produce 400 mL urine/day. A urine output of at least 0.5 mL/kg/hr indicates an adequate level of hydration and renal perfusion in adults with normal renal function.

- (2) Clinical manifestations of volume loss range from mild postural lightheadedness to resting tachycardia to a nondetectable blood pressure.
- (3) Flexibility in the body's handling of water intake is provided by two mechanisms.
 - (a) Antidiuretic hormone (ADH)
 - i. Increased body water \rightarrow decreased serum osmolality \rightarrow suppression of ADH secretion \rightarrow diuresis of free water
 - ii. Decreased free water \rightarrow increased serum osmolality \rightarrow stimulation of ADH release \rightarrow retention of free water
 - iii. Acute ($>8\%$ – 10%) volume depletion \rightarrow stimulation of ADH release \rightarrow retention of free water
 - (b) Aldosterone
 - i. Released by the adrenal gland in response to renin release by the kidney in low-volume states
 - ii. Stimulates retention of sodium by the renal tubules, which is followed by retention of water.
- c. Osmolality is determined by the number of particles of solute in relation to the amount of solvent. The most important solutes that contribute to serum osmolality are sodium, chloride, bicarbonate, and glucose. Large protein molecules do not make an important contribution, because they are small in number relative to these molecules.
 - (1) Serum osmolality is maintained primarily by regulation of ADH release by the pituitary.
 - (a) \uparrow osmolality $\rightarrow \uparrow$ ADH \rightarrow retention of free water
 - (b) \downarrow osmolality $\rightarrow \downarrow$ ADH \rightarrow excretion of free water
 - (2) Sodium regulation by aldosterone does not play a major role in the maintenance of normal serum osmolality.
 - (3) Clinical application
 - (a) D5W is roughly isotonic to plasma at the time of injection but, after the sugar is metabolized, only free water remains.
 - (b) 0.9% normal saline is isotonic to plasma; 0.45% normal saline is hypotonic to plasma.
 - i. Administering 1 L of normal saline provides 1 L of fluid into the extracellular space; of this, 250 mL (25%) remains in the intravascular space while 750 mL (75%) goes into the interstitial space.
 - ii. Administering 1 L of one-half normal saline provides 0.5 L of fluid into the extracellular space.
 - iii. Administering 1 L of D5W provides 333 mL into the extracellular space and 667 mL into the intracellular space; of this, only 83 mL (25%) remains in the intravascular space.

C. Sodium regulation

1. Facts

- a. Total body sodium = ~ 40 – 50 mEq/kg.
- b. Distribution of major cations and anions

- (1) Na^+ is the major extracellular cation (140 mEq/L)
 - (2) Cl^- and HCO_3^- are the major extracellular anions.
 - (3) K^+ and Mg^{++} are the major intracellular cations.
2. Concepts
- a. When physiologic changes cause intracellular-extracellular osmotic dysequilibrium, corrections are not made by movement of solute across the cell membrane, but rather, by movement of water. Disorders of sodium regulation actually represent a disorder of water regulation in which either too little or too much water is present in the body relative to solute. What this means is that hyponatremia and hypernatremia can occur with normal, low, or high total body sodium content.
 - b. There are three control points in water regulation (water conservation/excretion homeostasis).
 - (1) Hypothalamic thirst centers
 - (a) Increased by:
 - i. \downarrow Intracellular volume
 - ii. \downarrow Extracellular volume
 - iii. \uparrow Osmolality
 - iv. \uparrow Renin-angiotensin
 - v. \uparrow β -adrenergic stimulation
 - vi. Drugs (eg, lithium)
 - (b) Decreased by:
 - i. \uparrow Intracellular volume
 - ii. \uparrow Extracellular volume
 - iii. \downarrow Osmolality
 - (2) ADH synthesis and release
 - (a) Increased by:
 - i. \downarrow Effective arterial volume
 - ii. \uparrow Osmolality
 - iii. Drugs
 - (b) Decreased by:
 - i. \uparrow Effective arterial volume
 - ii. \downarrow Osmolality
 - iii. Drugs
 - (3) Kidney (renal tubule water absorption)
 - (a) Increased by:
 - i. \downarrow Glomerular filtration rate
 - ii. \uparrow Proximal tubule reabsorption
 - iii. \uparrow Loop reabsorption
 - iv. \uparrow Collecting tubule reabsorption
 - (b) Decreased by:
 - i. \downarrow Proximal tubule reabsorption
 - ii. \downarrow Loop reabsorption
 - iii. \downarrow Collecting tubule reabsorption

D. Potassium regulation

1. The normal serum concentration is 3.5–4.5 mEq/L.
 - a. Although the serum potassium is only 2% of total body potassium, in most settings it is a good indicator of total body stores.
 - b. Potassium is the primary intracellular cation.
 - c. An increased potassium concentration is a potent stimulus for aldosterone release.
2. Physiologic actions of potassium
 - a. Maintains resting membrane potential
 - b. Changes in the intracellular/extracellular gradient facilitate propagation of electrical impulses.
 - c. Filters freely through the glomerulus, is reabsorbed in the proximal and ascending tubules, and is then secreted into the distal tubule in exchange for sodium

E. Calcium regulation

1. Calcium is the most abundant cation in the body, but 99% is bound in bone; the rest is in the extracellular fluid, half of which is bound to plasma proteins and half as free (active) ions. The calcium gradient between extracellular and intracellular spaces is highly regulated at 10,000:1.
2. Total plasma calcium levels (8.5–10.5 mg/dL) are maintained by the function of vitamin D and parathyroid hormone.
 - a. Protein-bound = 4.0–4.5 mg/dL
 - b. Ionized = 4.2–4.8 mg/dL
 - c. A laboratory value reported in mEq/L = $\frac{1}{2}$ the amount in mg/dL, eg, 4.2 mg/dL = 2.1 mEq/L = 1.05 mOsm/L
3. Physiologic actions
 - a. Changes in H^+ concentration result in changes of ionized calcium because Ca^{++} binds to protein in place of H^+ . Decreased H^+ , such as in hyperventilation \rightarrow respiratory alkalosis \rightarrow \uparrow protein-bound calcium + \downarrow ionized serum Ca^{++} (relative hypocalcemia)
 - b. The ionized fraction of calcium mediates physiologic effects. Clinical effects of hypocalcemia are seen when the ionized levels fall below 3 mg/dL. Clinical manifestations of hypercalcemia are noted at total serum calcium levels >11 mg/dL.
 - (1) Serum albumin level (a decrease of 1 g/dL in albumin results in a decrease of 0.8 mg/dL in calcium) \rightarrow no change in ionized fraction
 - (2) Alkalosis (decreases the ionized fraction) \rightarrow no change in total serum calcium
 - (3) Acidosis (increases the ionized fraction) \rightarrow no change in total serum calcium
 - c. Calcium also plays a role in:
 - (1) Coagulation
 - (2) Platelet aggregation
 - (3) Hormone secretion
 - (4) Depolarization of electrical cells
 - (5) Functioning of contractile proteins
 - (6) Regulation of intracellular enzyme activity

F. Magnesium regulation

1. Magnesium is the second most abundant intracellular cation, and its regulation is closely related to calcium and phosphate.
2. Total body content is 24 g (2,000 mEq), 60% of which is in bone; most of the remaining 40% is intracellular. Magnesium is also an essential element in the production of cellular energy, cardiac excitability, the clotting mechanism, and in neuromuscular activity.
3. Dietary source is green vegetables; the usual daily requirement is ~25 mEq/day; normal serum level is 1.4–2.2 mEq/L.

G. Chloride regulation

1. Chloride is an extracellular anion that increases or decreases in concentration whenever changes occur in the concentrations of other anions, eg, metabolic acidosis (due to bicarbonate loss) results in an increase in chloride.
2. Functions of chloride
 - a. Acid-base balance
 - b. Osmotic balance
 - c. Water balance
3. A change in serum chloride is seldom a primary problem.

H. Physiologic "pumps" that maintain electrolyte balance

1. The sodium-potassium-ion pump, which is driven by ATP activity, is the major energy-using process in the body. Three sodium ions (3Na^+) are exchanged for two potassium ions (2K^+), thereby establishing an electrical gradient. This pump is directly inhibited by digoxin in the heart; this leads to increased intracellular concentration of calcium and increased contractility of heart muscle. Low potassium and high calcium levels potentiate this inhibition. This pump is responsible for:
 - a. Glucose uptake in the gut
 - b. Neurotransmitter uptake in the brain
 - c. Calcium transport in the heart
2. Calcium pumps also exist to keep calcium outside the cell.
3. A hydrogen-potassium exchange may also occur. The classic teaching is that when metabolic acidosis is caused by organic acids that do not freely cross the cell membrane (eg, ketoacids), H^+ moves inside the cell to be buffered and is exchanged for K^+ . However, while hyperkalemia is commonly associated with diabetic ketoacidosis, it is not commonly seen in alcoholic ketoacidosis, suggesting that insulin may have a significant effect on potassium regulation. Increases in serum K^+ do not occur when the metabolic acidosis is caused by other organic ions (such as lactate).

I. The anion gap

1. The anion gap is the difference between serum anions and cations. In a physiologically normal person, measured serum anions are Cl^- and HCO_3^- and unmeasured anions are the negative charges on serum albumin, phosphate, and sulfate. Measured serum cations are Na^+ , K^+ , Mg^{++} , and Ca^{++} . In the clinical setting, the anion gap is calculated from the predominant measured cation (Na^+) and anions (Cl^- and HCO_3^-).

$$\text{Anion gap} = \text{Na}^+ - (\text{Cl}^- \text{ and } \text{HCO}_3^-)$$

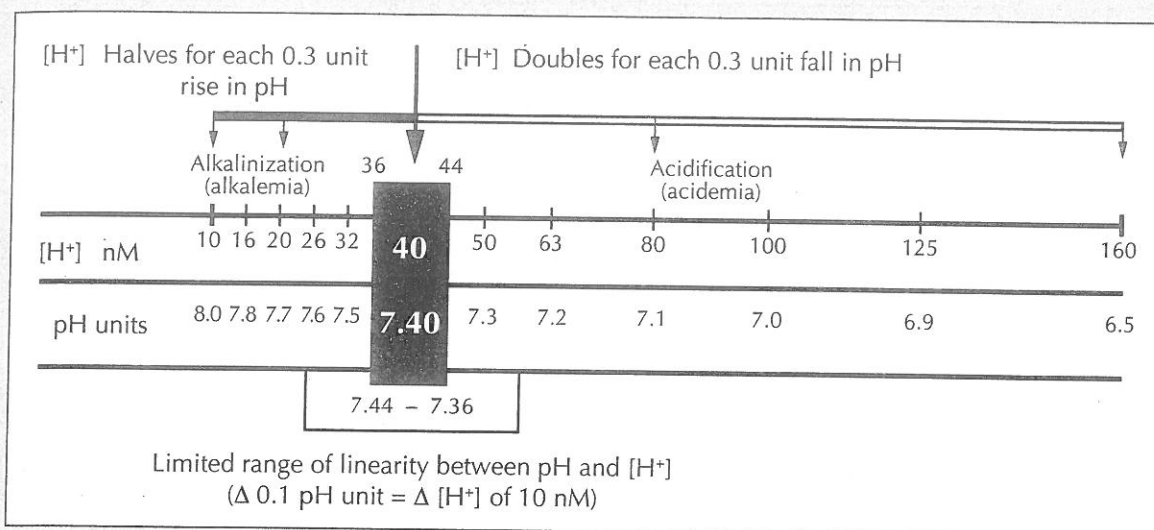
$$\text{Normal range} = 8\text{--}16 \text{ mEq}$$
2. A significantly increased anion gap signifies the presence of excessive unmeasured anions (usually due to metabolic acidosis). A small or negative anion gap is seen when there is:

- a. An excess of unmeasured serum cations *or*
- b. An increase in unmeasured positively charged proteins *or*
- c. A decrease in serum albumin *or*
- d. An increase in negatively charged particles that are mistaken for chloride (such as bromide or iodide)
- 3. Increased cations are seen in the presence of lithium and myeloma proteins. Starvation leads to a decrease in the unmeasured negative charges on serum albumin and a decrease in the anion gap. Iodide and bromide are mistaken for chloride by most laboratories, so that poisoning with these agents lowers the calculated anion gap. Triglyceride levels >600 mg/dL may also cause chloride levels to be overestimated.
- 4. Causes of anion gap acidosis
 - a. Diabetic ketoacidosis
 - b. Alcoholic ketoacidosis
 - c. Lactic acidosis
 - d. Renal failure (uremia)
 - e. Certain drugs or toxins
 - (1) Salicylates
 - (2) Paraldehyde, metformin, isoniazid, iron
 - (3) Methanol
 - (4) Ethylene glycol
 - (5) Nucleoside analogue reverse-transcriptase inhibitors (eg, zidovudine)
- 5. If an abnormally high anion gap occurs in the presence of an abnormally high osmolal gap (>10 mOsm difference between the measured and calculated serum osmolality), ingestion of methanol or ethylene glycol should be suspected.
- 6. **If an abnormally high osmolal gap is seen in the presence of a normal anion gap, suspect the presence of one of the following:**
 - a. **Ethanol**
 - b. **Isopropyl alcohol**
 - c. **Glycerol**
 - d. **Sorbitol**
 - e. **Mannitol**
 - f. **Acetone**

J. Pathophysiology of acid-base disorders

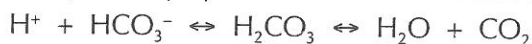
- 1. The body normally maintains a critical acid-base balance primarily by handling three types of acids:
 - a. Fixed acids (H^+), which are normally produced from dietary intake (sulfates and phosphates)
 - b. Acids produced from pathologic processes and abnormal metabolic pathways
 - c. Exogenous acids (toxins, medications, iatrogenic administration)
- 2. Approximately 15,000 mg of volatile acid (CO_2) is excreted from the lungs each day.
- 3. Approximately 70 mEq/L of nonvolatile acids are produced each day.
- 4. There are three mechanisms for renal excretion of these acids:
 - a. Direct hydrogen excretion (~0.1 mEq/day)

- b. Excretion of urinary buffers (~20 mEq/day)
- c. Excretion with ammonia (~50 mEq/day)
5. Hydrogen ion activity is clinically reported as pH, which is the negative logarithm of the hydrogen ion concentration. At normal body temperature, a system of buffers maintains the pH of blood at 7.4 (40 nM H^+ concentration).



Relationship of pH to H⁺ Concentration

6. Minute-to-minute regulation of hydrogen ion concentration in the blood and interstitium is maintained primarily by the bicarbonate-carbonic acid system:



To maintain this equilibrium, reciprocal changes occur when a component of this equation is "out of balance."



7. The relationship between pH, bicarbonate, and CO₂ described by the Henderson-Hasselbach equation is always met:

$$H^+ = 24 \times \frac{PCO_2}{HCO_3^-}$$

Therefore, it can be used to verify the reliability of laboratory determinations (pH, CO₂, HCO₃⁻) or to predict any one value (given two values).

8. When the balance between bicarbonate and carbonic acid is disrupted, compensatory mechanisms are stimulated. Metabolic compensation occurs in response to respiratory abnormalities, and respiratory compensation occurs in response to metabolic abnormalities. The following parameters can be used to predict the expected compensatory response **with acute changes in the $p\text{CO}_2$** :
 - $\text{pH} \downarrow$ by 0.08 units for each 10 mmHg \uparrow in CO_2
 - $\text{pH} \uparrow$ by 0.08 units for each 10 mmHg \downarrow in CO_2
 - a. Metabolic compensation for respiratory acidosis is facilitated by increased retention of bicarbonate by the kidney. In respiratory alkalosis, the kidney secretes bicarbonate.
 - b. Respiratory compensation for metabolic alkalosis is limited by the development of hypoxemia that occurs during hypoventilation.
 - c. Respiratory compensation for metabolic acidosis is rapid. Experimental observation of respiratory compensation for uncomplicated metabolic acidosis has determined the following:
 - (1) The pH is never compensated to a normal level (>7.35).
 - (2) The $p\text{CO}_2$ almost never goes lower than 10.
 - (3) The $p\text{CO}_2$ approximates $1.5 \times \text{bicarb} + 8 \pm 2$.
9. Lactate is the reduced form of pyruvate. Serum lactate level is determined by three factors:
 - a. Pyruvate (\uparrow with glycolysis and \downarrow with gluconeogenesis)
 - b. Intracellular redox state (NADH/NAD), which is O_2 -dependent
 - c. Intracellular pH

"Lactic acidosis" occurs when tissue hypoxia results in a shift to anaerobic metabolism, described as the inability to dump electrons into oxygen (via oxidative phosphorylation); this causes an increase in the reducing capacity of cells (that is reflected by an increased NADH/NAD) and an increased lactate to pyruvate ratio: hypoxemia $\rightarrow \uparrow \text{NADH/NAD} \rightarrow \uparrow \text{lactate/pyruvate}$. The actual cause of the metabolic acidosis seen in patients with lactic acidosis is not lactic acid, but the accumulated protons that do not combine with ADP to form ATP in oxidative phosphorylation; this is the reason that the measurements of serum lactate often do not correlate with the anion gap.

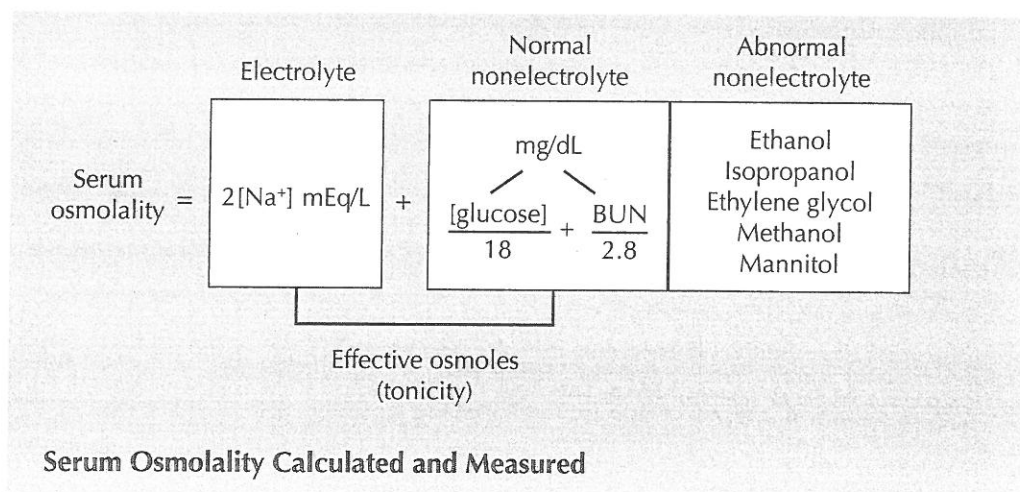
II. ELECTROLYTE DISORDERS

A. Hyponatremia

1. Definition: serum $\text{Na}^+ < 135 \text{ mEq/L}$ ($< 120 \text{ mEq/L}$ = severe)
2. Classic clinical scenario
 - a. Clinical presentation: headache, confusion, nausea, vomiting, muscle cramps, and seizures; may also be asymptomatic
 - b. There are no classic ECG findings.
 - c. Etiology
 - (1) Hypervolemic: increased prerenal sodium and water resorption \rightarrow inability to deliver free water to the distal nephron (CHF, cirrhosis)
 - (2) Hypovolemic: excessive sodium losses (vomiting, diarrhea, nephrosis, diuretics, osmotic diuresis, adrenocortical insufficiency)

- (3) Psychogenic polydipsia: excessive intake of water
 - (4) Normovolemic: secretion of inappropriate antidiuretic hormone (SIADH)
 - (5) Factitious or pseudohyponatremia: hyperglycemia, multiple myeloma, or hyperlipidemia; for every 100mg% increase in serum glucose, the apparent measured serum sodium is decreased by 1.6–1.8 mEq/L per 100 mg/dL.
- d. Acute treatment
- (1) Indicated only if the patient is symptomatic and the duration of hyponatremia is <24 hours
 - (2) **Central pontine myelinolysis—rapid correction of hyponatremia has been associated with the development of:**
 - (a) Neurologic deterioration associated with a rate of correction >0.6 mEq/L/hr or >25 mEq/48 hours in cases of hyponatremia that has been present >2 days.
 - (b) It has been associated with correction rates >2.5 mEq/hr when hyponatremia has been present <2 days.
 - (c) There is no evidence that correction of very acute (<24 hours) hyponatremia is harmful. When correction is needed, estimation of the sodium deficit may be useful.

$\text{Na}^+ \text{ needed (in mEq total)} = 0.6 (\% \text{ total body water}) \times \text{weight (kg)} \times 140 - \text{actual serum Na}^+ \text{ in mEq/L}$
- e. Clinical approach to the assessment of hyponatremia
- (1) Ask yourself, "Is this an emergency?"
 - (a) Rate of development of symptoms ("yes" if <24 hours)
 - (b) Altered status or seizures ("yes" if present)
 - (2) Is the cause of hyponatremia evident on the basis of history and physical examination? If not, measurement of the serum osmolality will be very helpful.



Normal: calculated Osm – 10 mOsm/L = measured Osm (Na^+ , glucose, BUN)

Osmolal gap: calculated Osm < measured Osm (nonelectrolyte solute other than glucose or urea, or pseudohyponatremia)

(3) Categorize the hyponatremic patient into one of two groups, which will guide therapy (normal or high osmolality, low osmolality).

(a) Pseudohyponatremia

i. If the measured osmolality is normal and the calculated osmolality is low:

- Cause: low water volume with high solute volume; the sodium concentration is normal in the aqueous phase.
- Differential diagnosis: multiple myeloma, hyperlipidemia
- Treatment: none

ii. If the measured osmolality is high and the calculated osmolality is normal to high:

- Cause: free water moves from the intracellular space to the high solute concentration in the extracellular space.
- Differential diagnosis: hyperglycemia, mannitol, glycerol excess
- Treatment
 - Hyperglycemia → insulin and saline
 - Mannitol → saline

(b) Low osmolality (most common)

i.
$$\left[\begin{array}{l} \text{measured osmolality} = \text{low} \\ \text{calculated osmolality} = \text{low} \end{array} > \text{both equal} \right]$$

ii. The causes are classified and the treatments are based on evaluation of two parameters:

- Volume status of the patient (hypovolemic, euvolemic, hypervolemic)
- Measurement of urine sodium

iii. Hypovolemic hypo-osmolar hyponatremia

- Contracted effective intravascular volume → kidney avidly reabsorbs water → hyponatremia
- Clinical examination reflects the presence of hypovolemia: postural hypotension, poor skin turgor, flat neck veins
- Measurement of urinary sodium can help determine if the volume depletion is due to a renal or extrarenal source.
 - Renal sodium loss (urine Na^+ >20 mEq/L)
 - Diuretics
 - Mineralocorticoid deficiency
 - Salt-wasting nephropathy
 - Osmotic diuresis (hyperglycemia, mannitol)
 - Extrarenal sodium loss (urine Na^+ <10 mEq/L)
 - Excessive sweating
 - Vomiting
 - Diarrhea
 - Fistula
 - “Third space” burns, hemorrhagic pancreatitis, peritonitis
 - Traumatized muscle
- Treatment: isotonic saline (regardless of the cause)

(c) Euvolemic hyposmolar hyponatremia

- i. The volume status is clinically normal or there is evidence of mild extracellular volume excess.
- ii. Etiology
 - Endocrine (hypothyroidism, glucocorticoid deficiency, SIADH)
 - Nonendocrine (pain, drugs, psychogenic water drinker)
- iii. Measurement of urinary sodium is usually high (>20 mEq/L).
- iv. The diagnosis of SIADH is one of exclusion.
 - Typical laboratory data: \downarrow BUN, \uparrow urine osmolality (>150 mOsm), \uparrow urine Na^+ (>20 mEq/L)
 - Treatment is based on the severity of the disorder. For acute, severe, symptomatic hyponatremia, hypertonic saline or furosemide plus saline may be indicated for rapid correction. For less severe cases, the restriction of free water is appropriate therapy.

(d) Hypervolemic hypo-osmolar hyponatremia (most common form)

- i. Volume status: clinically edematous (the extracellular volume is expanded but "ineffective"; the kidney perceives a low intravascular [extracellular] volume and resorbs excess salt and water; as a result, there is an excess of total body sodium and a greater excess of total body water)
- ii. Measurement of urinary sodium
 - >20 mEq/L (renal): acute or chronic renal failure
 - <10 mEq/L (extrarenal): nephrotic syndrome, hepatic cirrhosis, CHF
- iii. Management
 - Treat underlying disorder
 - Water restriction
 - If hyponatremia is severe: loop diuretics followed by hypertonic saline
 - Anuric patients: dialysis is usually indicated.

B. Hypernatremia

1. Definition: serum Na^+ >145 mEq/L (>155 mEq/L = severe)
2. Classic clinical scenario
 - a. Clinical presentation: \downarrow level of consciousness, dehydrated, \pm seizures
 - b. There are no classic ECG findings.
 - c. Etiology: sodium and water loss
 - (1) \uparrow water loss (hyperpnea, excessive sweating, vomiting)
 - (2) \uparrow diuresis (diabetes insipidus, acute tubular necrosis, diuretics, and postobstructive diuresis)
 - d. Hypernatremic states can be categorized into one of two groups:
 - (1) Water loss (\downarrow intake or \uparrow output)
 - (a) Patients who have a low or normal total body sodium
 - i. Reduced water intake
 - ii. Abnormal thirst mechanism
 - iii. Inability to drink (physical, neurologic)

- iv. Osmotic diuresis
 - Diabetic ketoacidosis
 - Hyperosmolar nonketotic coma
- (b) Patients who have abnormalities in production of, or renal response to, ADH
 - i. Diabetes insipidus (central, nephrogenic)
 - ii. Drugs (lithium, aminoglycosides, phenytoin, fluoride)
- (2) Sodium gain (iatrogenic)—these patients have increased total body sodium.
 - (a) Saline administration (including hypertonic saline)
 - (b) Hypertonic dialysis
 - (c) Hypertonic feedings
 - (d) "Bicarb" administration
- e. Management
 - (1) Guided by the rate of development of the hypernatremia, the presence of symptoms, and the absolute sodium level
 - (2) Calculate the water deficit

$$\text{water deficit (L)} = \left(\frac{\text{measured } [\text{Na}^+]}{\text{normal } [\text{Na}^+]} \right) - 1$$

- (3) When initiating therapy in patients with severe water loss and hemodynamic compromise, start with isotonic saline (0.9% normal saline).
- (4) In patients with increased total body sodium due to sodium gain, it may be necessary to use a loop diuretic in conjunction with D5W. In severely ill patients, hemodialysis may be lifesaving.
- (5) Patients with prolonged hypernatremia have normal intracellular volume due to the acquisition of "idiogenic osmoles" that equalize intracellular and extracellular osmolarities. The rapid correction of hypernatremia may lead to excessive movement of water into these hyperosmolar cells, resulting in cerebral edema. Therefore, sodium levels should not be lowered faster than 10–15 mEq/L/day; only half of the water deficit should be corrected in the first 24 hours; in most clinical situations, the total deficit will require replacement over the next 24–48 hours so that the serum osmolality drops about 2 mOsm/kg/hr.

C. Dehydration

- 1. Three categories: hypotonic, isotonic, and hypertonic (Following is a summary of the information presented above on hypo- and-hypernatremia, which is important to understand the cause and type of dehydration in a particular patient.)
- 2. Hypotonic dehydration
 - a. Loss of sodium in excess of water
 - b. Serum Na^+ is low.
 - c. Differential diagnoses
 - (1) Diuretic excess
 - (2) "Fluid space" losses
 - (3) Vomiting
 - (4) Adrenocortical insufficiency
- 3. Isotonic dehydration

- a. Loss of sodium = loss of water
- b. Serum Na^+ is normal.
- c. Most commonly associated clinical condition is vomiting.
- 4. Hypertonic dehydration
 - a. Loss of water in excess of sodium
 - b. Serum Na^+ is increased.
 - c. Differential diagnoses
 - (1) Diarrhea
 - (2) Lack of access to water
 - (3) Nonketotic hyperosmolar coma
 - (4) Diabetes insipidus

D. Hypokalemia

- 1. Definition: serum $\text{K}^+ < 3.5 \text{ mEq/L}$ ($<2.5 \text{ mEq/L}$ = severe)
- 2. Classic clinical scenario
 - a. Muscle weakness and ECG changes (flat T waves and presence of U waves); clinical findings may be minimal.
 - b. Etiology: excess loss
 - (1) GI conditions: vomiting, gastric suctioning, starvation, chronic diarrhea, chronic laxative abuse, villous adenoma, colon cancer, licorice ingestion
 - (2) Renal conditions with metabolic acidosis
 - (a) Renal tubular acidosis
 - (b) Postobstructive diuresis
 - (3) Renal conditions with metabolic alkalosis
 - (a) Diuretics
 - (b) Cushing syndrome
 - (4) Familial periodic paralysis
 - (5) Intracellular shifts of glucose/insulin
 - (6) Transcellular shifts due to larger doses of β -agonists
 - (7) Excess mineralocorticoids (\uparrow renal excretion of K^+)
 - (8) Sweating
 - (9) Hyperthyroidism
 - (10) Hypomagnesemia
 - c. Clinical presentation of severe hypokalemia ($<2.5 \text{ mEq/L}$)
 - (1) Neuromuscular
 - (a) Pronounced weakness
 - (b) Hyporeflexia
 - (c) Paralysis
 - (2) Cardiac (Hypokalemia enhances digitalis toxicity even with a normal maintenance dose.)
 - (a) Bradycardia/sinus arrest, first-degree AV block, idioventricular rhythm, ventricular tachycardia, ventricular fibrillation, ventricular standstill
 - (b) ECG changes

- i. U waves
 - ii. Flat T waves
 - iii. ST depression (present when hypokalemia is severe)
 - iv. Prolonged QT interval (present when hypokalemia is severe)
- (3) GI → ileus
- d. Management
 - (1) Subacute or chronic hypokalemia (the most common and not usually life threatening)
 - (a) Approximately 150 mEq are needed to raise the serum K^+ level by 1 mEq/L in subacute or chronic deficiency states.
 - (b) Estimate a 150–200 mEq deficit for each mEq/L below 3.5 in these situations.
 - (c) Replace the potassium deficit orally (unless the patient has signs and symptoms of severe hypokalemia); also replace any associated magnesium deficit.
 - (2) Acute hypokalemia (less common and usually life threatening)
 - (a) Approximately 40 mEq are needed to raise the serum level 1 mEq/L.
 - (b) Estimate a deficit of 40 mEq for each mEq/L below 3.5 in this situation.
 - (c) No more than 80 mEq should be placed in 1 L of IV fluid, and no more than 40 mEq should be given over 1 hour in most cases.
 - (d) Patients receiving potassium at >20 mEq/hr as an IV infusion need to be observed in an intensive care setting on a cardiac monitor.
 - (e) Patients who present with hypokalemia in the setting of diabetic ketoacidosis may develop life-threatening hypokalemia as therapy of the diabetic ketoacidosis is instituted. These patients may require potassium replacement at rates as high as 60 mEq/hr.

E. Hyperkalemia

1. Definition: serum $K^+ > 4.5$ mEq/L (>6.5 mEq/L = severe)
2. Pathophysiology
 - a. Acidosis: renal failure, diabetic ketoacidosis
 - b. Cell lysis: chemotherapy, hemolysis, burns, rhabdomyolysis
 - c. Mineralocorticoids (Addison disease)
 - d. Medications (triamterene, spironolactone, ACE inhibitors, NSAIDs, β -blockers)
3. Clinical presentation
 - a. Signs and symptoms are multiple and often difficult to discern from those of the primary illness or condition that precipitated the hyperkalemia.
 - b. Neuromuscular findings may begin with lethargy and weakness and progress to paralysis and areflexia.
 - c. Cardiac findings
 - (1) Hypotension
 - (2) Dysrhythmias
 - (3) ECG changes progress as severity of hyperkalemia progresses: tall peaked T waves → wide QRS complexes → sine waves; however, ECG findings may be minimal, especially in renal patients.

4. Etiology

a. Factitious

- (1) Thrombocytosis
- (2) Leukocytosis
- (3) Prolonged tourniquet time
- (4) In-vitro hemolysis

b. Decreased renal excretion

- (1) Renal insufficiency
- (2) Adrenal or aldosterone insufficiency
- (3) Drugs (eg, potassium-sparing diuretics, ACE inhibitors)
- (4) Type IV renal tubular acidosis (diabetes)

c. Increased potassium load

- (1) Cellular breakdown (trauma, tumor lysis, rhabdomyolysis)
- (2) Potassium-containing salt substitutes
- (3) Hemolysis
- (4) GI bleeding
- (5) High-dose penicillin VK

d. Decreased cellular uptake of potassium

- (1) Diabetic ketoacidosis (serum K^+ level increases 0.6 mEq/L for every 0.1 decrease in pH)
- (2) Drugs (β -blocker or digoxin overdose, succinylcholine)

5. Management

- a. If there are no ECG signs, treatment can be restricted to methods that increase potassium excretion through the bowel or kidneys or decrease potassium intake (diet).
- b. Because the kidney is the major determinant of the serum potassium concentration, emergent treatment of hyperkalemia requires a method that either blocks the site of action of the excess potassium or shifts the excess potassium from one compartment to another. Hyperkalemia due to familial periodic paralysis is caused by potassium shifts (not total body depletion); therefore, overcorrection may occur in this setting.
 - (1) Calcium chloride or gluconate antagonizes the effects of potassium in the myocardium, thus decreasing membrane irritability, which reduces the risk of developing a ventricular dysrhythmia.
 - (2) Glucose and insulin, bicarbonate, and β -adrenergic agonists redistribute excess potassium from the extracellular to the intracellular compartment.
 - (3) Therapy with dialysis or an exchange resin (polystyrene sulfonate) should be started shortly after using any emergent treatment.

Table 32: Treatments for Hyperkalemia

Treatment	Mechanism	Dosage	Onset	Duration of Effect
Calcium gluconate (10%)	Membrane stabilization	10–20 mL IV	1–3 min	20–50 min
Sodium bicarbonate	Redistribution of K ⁺	1 mEq/kg IV	5–10 min	1–2 hours
Albuterol	Redistribution of K ⁺	10–20 mg by inhaler	30 min	2–4 hours
Insulin plus glucose	Redistribution	5–10 units of regular insulin with 1–2 ampules D50 IV	30 min	4–6 hours
Cation exchange resin (sodium polystyrene)	Excretion	25–50 g orally or per rectum with sorbitol	1–2 hours	4–6 hours
Peritoneal or hemodialysis	Excretion	—	Within minutes after starting	Until dialysis is completed
Diuretics	Excretion			
Furosemide		40 mg IV	With start of diuresis	Until diuresis ends

F. Hypocalcemia

1. Definition: serum Ca⁺⁺ <8.5 mg/dL (<7 mg/dL = severe)
2. Etiology
 - a. Renal failure
 - b. Hypoparathyroidism
 - c. Acute pancreatitis
 - d. GI loss: chronic diarrhea, malabsorption
3. Clinical manifestations of hypocalcemia usually occur when the serum calcium level falls below 6.5 mg/dL. Other signs and symptoms include:
 - a. Neuromuscular
 - (1) Paresthesias (perioral and peripheral)
 - (2) Carpopedal spasms
 - (3) Chvostek or Trousseau sign
 - (4) Hyperreflexia
 - (5) Seizures
 - b. Cardiovascular
 - (1) Hypotension
 - (2) CHF
 - (3) Dysrhythmias
 - (4) Prolonged QT interval
4. Although hypocalcemia is uncommon, when it does occur, common causes include the following:
 - a. Acute pancreatitis with extensive fat necrosis (fat binds calcium)
 - b. Impaired absorption of vitamin D (malabsorption) or impaired production of 1,25-dihydroxy vitamin D (renal or hepatic failure, anticonvulsant therapy)

- c. Hypomagnesemia (makes end-organs resistant to parathyroid hormone); common in alcoholics
 - d. Hypoparathyroidism
 - e. Drugs (very rare)
 - (1) Phenytoin, phenobarbital
 - (2) Aminoglycosides
 - (3) Steroids
 - (4) Loop diuretics
 - f. Alkalosis ($0.1 \uparrow$ in pH, $0.05\text{--}0.08 \text{ mEq/L} \downarrow$ in Ca^{++})
 - (1) Respiratory \rightarrow hyperventilation
 - (2) Metabolic \rightarrow vomiting, diarrhea, malabsorption
 - g. Rapid and massive blood transfusions
 - h. Rhabdomyolysis (increased calcium binding to injured tissue)
 - i. Sepsis and shock states
 - j. Renal failure
5. Management
- a. The ultimate goal of therapy is treatment of the underlying cause.
 - b. Emergent treatment with IV replacement is indicated if the patient is acutely symptomatic: 10 mL CaCl_2 (10% solution) IV push over 15–20 minutes (repeat as needed).
 - c. Transfusion of citrated blood may result in hypocalcemia (which is transient) and does not require routine treatment unless signs of hyperreflexia develop.

G. Hypercalcemia

- 1. Definition: serum $\text{Ca}^{++} > 0.5 \text{ mg/dL}$ ($>12 \text{ mg/dL}$ = severe)
- 2. Clinical presentation: "groans, stones, psych overtones"
 - a. Neuromuscular
 - (1) Weakness, hyporeflexia, hypotonia
 - (2) Apathy, confusion, depression
 - (3) Lethargy, stupor, coma (if the hypercalcemia is the cause of the coma, serum Ca^{++} is $>13.5 \text{ mg}$)
 - b. Cardiovascular
 - (1) Hypertension
 - (2) Dysrhythmias
 - (3) ECG abnormalities
 - (a) Short QT interval
 - (b) Coving of the ST segment and T wave
 - (c) Widening of the T wave
 - (4) Digitalis sensitivity (hypercalcemia enhances digitalis toxicity)
 - c. GI
 - (1) Anorexia
 - (2) Nausea, vomiting
 - (3) Abdominal pain
 - (4) Constipation

- d. Renal
 - (1) Nephrolithiasis
 - (2) Renal failure
 - (3) Polyuria
- 3. Etiology
 - a. Malignancy
 - (1) Hematologic (Hodgkin, lymphomas, multiple myeloma)
 - (2) Metastasis to bone (thyroid, lung, breast, kidney); a serum Ca^{++} >13.5 mg/dL suggests this diagnosis.
 - (3) Parathyroid hormone-producing lung/kidney cancers
 - b. Excess intake
 - (1) Milk-alkali syndrome
 - (2) Vitamin D (or vitamin A) toxicity
 - c. Acute osteoporosis
 - (1) Immobilization of young patients
 - (2) Paget disease (especially with immobilization)
 - d. Hyperparathyroidism (a serum Ca^{++} >11 mg/dL suggests this diagnosis)
 - e. Drugs
 - (1) Lithium
 - (2) Thiazide diuretics
- 4. Management
 - a. Initiate for symptomatic patients or for Ca^{++} >14 mg/dL regardless of symptoms
 - b. Saline hydration
 - c. Furosemide 1 mg/kg as needed for fluid overload (thiazide diuretics contraindicated in patients with hypercalcemia)
 - d. Potassium replacement as needed
 - e. Hydrocortisone
 - f. Drugs that decrease bone absorption
 - (1) Calcitonin
 - (2) Glucocorticoids
 - (3) Bisphosphonates
 - (4) Gallium nitrate
 - g. Treatment of underlying neoplasm if present

H. Hypomagnesemia

- 1. Definition: serum Mg^{++} <1.4 mEq/L (<0.5 mEq/L = severe)
- 2. Clinical presentation: remember that secondary hypocalcemia occurs with hypomagnesemia, so signs and symptoms of hypocalcemia are also present.
 - a. Neuromuscular
 - (1) Irritability/lethargy
 - (2) Tetany
 - (3) Tremor

- (4) Carpopedal spasm
 - (5) Hyperreflexia
 - b. Cardiovascular
 - (1) Hypotension
 - (2) Dysrhythmias (premature ventricular contractions, ventricular tachycardia, ventricular fibrillation): may potentiate digitalis toxicity
 - (3) ECG findings
 - (a) Prolonged PR and QT intervals
 - (b) Wide QRS complex
 - (c) ST depression
 - (d) Broad flat T waves with precordial T wave inversion
 - c. Metabolic
 - (1) Hypokalemia
 - (2) Hypocalcemia
3. Etiology
- a. Inadequate intake or absorption
 - (1) Malnutrition
 - (2) IV hyperalimentation (inadequate magnesium supplementation)
 - (3) Malabsorption
 - (a) Primary malabsorption syndrome
 - (b) Chronic diarrhea
 - (c) Fistulas
 - (d) Abdominal radiation
 - b. Endocrine disorders
 - (1) Diabetic ketoacidosis
 - (2) Hyperparathyroidism
 - (3) Hyperaldosteronism
 - c. Alcoholism (chronic)
 - d. Pancreatitis
 - e. Drugs
 - (1) Aminoglycosides
 - (2) Amphotericin B
 - (3) Diuretics
 - (4) β -agonists
 - (5) Cyclosporine
4. Management
- a. Check for hypocalcemia, hypokalemia.
 - b. Treat the underlying cause.
 - c. Replacement therapy
 - (1) For life-threatening problems (dysrhythmias, seizures): 1–2 g 10% MgSO_4 IV in 1–5 minutes, followed by an infusion of 1–2 g/hr.
 - (2) For less urgent presentations: 1–2 g 10% MgSO_4 IV over 3 hours or 2 g of a 50% solution IM every 4 hours \times 5 doses

I. Hypermagnesemia

1. Definition: serum Mg^{++} >2.2 mEq/L (>3 mEq/L = severe)
2. Clinical presentation (usually does not occur until the serum level is >3 mEq/L)
 - a. Neuromuscular
 - (1) Weakness
 - (2) Drowsiness/lethargy
 - (3) Slurred speech
 - (4) Hyporeflexia
 - (5) Coma and respiratory failure (serum Mg^{++} >5 mEq/L)
 - b. Cardiovascular
 - (1) Bradycardia \rightarrow AV block \rightarrow asystole
 - (2) Prolonged PR and QT intervals with extreme elevations of ST segments and T waves
 - (3) Vasodilation and hypotension (serum Mg^{++} >10 – 12 mEq/L)
 - c. Because magnesium is used to treat some clinical situations (eg, preterm labor), recognizing these findings is important in case of iatrogenic hypermagnesemia.
3. Etiology
 - a. Renal failure (particularly in patients who were given magnesium-containing drugs)
 - b. Iatrogenic: preeclamptic and eclamptic patients who were treated with $MgSO_4$ to reduce blood pressure and/or control seizure activity
 - c. Untreated diabetic ketoacidosis
 - d. Adrenal insufficiency
 - e. Rhabdomyolysis
4. Management
 - a. Discontinue exogenous magnesium
 - b. Diuretics
 - c. Calcium
 - d. Dialysis

J. Hypochloremia

1. Definition: serum Cl^- <100 mEq/L (<70 mEq/L = severe)
2. Etiology
 - a. Hypokalemic alkalosis (need to replace Na^+ , K^+ , and Cl^-)
 - b. GI losses
 - (1) Vomiting (including pyloric stenosis)
 - (2) Diarrhea (including ulcerative colitis)
 - c. Heat exhaustion
 - d. Acute infections such as pneumonia (cause unknown)
3. Management
 - a. Treat underlying disorder
 - b. Chloride replacement with NaCl is indicated if hypochloremia is severe or associated with hypokalemic alkalosis.

K. Hyperchloremia

1. Definition: serum $\text{Cl}^- > 110 \text{ mEq/L}$ ($> 120 \text{ mEq/L}$ = severe)
2. Etiology
 - a. Dehydration
 - b. Cardiac decompensation
 - c. Bicarbonate loss (GI, renal)
3. Management
 - a. For GI bicarbonate loss \rightarrow normal saline
 - b. For renal bicarbonate loss \rightarrow oral bicarbonate and renal consultation

III. ACID-BASE DISORDERS**A. General approach to the patient with an acid-base abnormality**

1. Basic definitions
 - a. -osis: refers to a process
 - b. -emia: refers to H^+ concentration in the blood
 - c. alkalosis: a process leading to alkalemia
 - d. acidosis: a process leading to acidemia
 - e. alkalemia: $\text{pH} > 7.44$ or $[\text{H}^+] < 36 \text{ mEq/L}$
 - f. acidemia: $\text{pH} < 7.36$ or $[\text{H}^+] > 44 \text{ mEq/L}$
 - g. metabolic disorders: changes in HCO_3^-
 - h. respiratory disorders: changes in pCO_2
2. What to do with abnormal arterial blood gases
 - a. The history and physical examination are important; pay particular attention to the following:
 - (1) Volume status (state of hydration)
 - (2) Respiratory status (rate, pattern)
 - (3) Known medical problems
 - (4) Known medications or illicit drug use (and consider their toxic effects)
 - (a) Opioids (hypoventilation \rightarrow respiratory acidosis)
 - (b) Salicylism (hyperventilation \rightarrow respiratory alkalosis)
 - (c) Toxic alcohols (metabolic acidosis)
 - b. Look at the arterial blood gases and electrolytes together. Determine the anion gap and classify the disturbance as acidosis or alkalosis (compensation will not overcorrect). Then determine if the cause is metabolic or respiratory. Then look at the extent and effectiveness of compensation. This can help judge acuteness.
 - (1) Respiratory alkalosis (decreased pCO_2)
 - (a) Toxic: salicylates, sympathomimetic agents
 - (b) Nontoxic: increased intracranial pressure, liver failure, hypoxia, heart failure, sepsis, pulmonary embolism, psychogenic
 - (2) Respiratory acidosis (increased pCO_2)
 - (a) Toxic: sedative hypnotics, opioids

- (b) Nontoxic: other causes of respiratory failure
 - (3) Metabolic alkalosis (increased bicarbonate): volume depletion (any cause), Barter syndrome, hyperaldosteronism
 - (4) Anion-gap metabolic acidosis (decreased bicarbonate)
 - (a) Toxic: salicylates, phenformin, carbon monoxide, cyanide, isoniazid (seizures), iron, methanol, ethylene glycol
 - (b) Nontoxic: seizures, shock, hypoxia, sepsis, ketoacidosis, uremia
 - (5) Nonanion gap metabolic acidosis (decreased bicarbonate, increased chloride)
 - (a) Toxic: chronic toluene exposure
 - (b) Nontoxic: bicarbonate wasting conditions such as renal tubular acidosis or diarrhea, ketone-wasting
 - (6) Mixed disorder
 - (7) Remember: "Normal is not normal when it should be abnormal." For example, a person breathing 40 times a minute should not have a $p\text{CO}_2$ of 40. Even though this is a "normal" value, this is a sign of impending respiratory failure.
- c. If an anion gap is present, pinpoint the problem (see metabolic acidosis, page 779).
 - (1) Consider causes (measurement of serum ketones, lactate, or glucose).
 - (2) Consider an ethylene glycol, methanol, or salicylate level.
 - (3) Consider causes of an anion gap other than metabolic acidosis.
 - (a) Severe metabolic alkalosis
 - (b) Anion infusion
 - (c) Antibiotics
 - (4) Causes of a decreased anion gap
 - (a) Hypoalbuminemia
 - (b) Bromide or iodide poisoning
 - (c) Multiple myeloma
 - (5) Measure the osmol gap (the difference between the measured and calculated osmolality).
 - (a) The presence of an osmol gap suggests the laboratory is measuring an osmotically active substance that you are not using in your calculation.
 - (b) Common causes of an osmol gap
 - i. Ethanol
 - ii. Methanol
 - iii. Ethylene glycol
 - iv. Isopropanol
 - v. Acetone
 - vi. Glycerol
 - vii. Mannitol
 - viii. Uremia
 - ix. Ketoacidosis
 - (c) Of the alcohols, ethanol and isopropanol have an osmolal gap but a normal anion gap.
 - (d) In uremia and ketoacidosis, the osmolal gap is usually <20 .

- d. Questions to answer to determine if a mixed disorder is present:
- (1) Do the clinical rules for "pure metabolic acidosis" apply? Deviation implies the presence of a mixed disorder.
 - (a) Respiratory compensation should not raise the pH to normal (>7.35).
 - (b) The $p\text{CO}_2$ approximates $1.5 \times \text{bicarbonate} + 8 \pm 2$
 - (c) For a pure anion gap acidosis, the increase in the anion gap should equal the decrease in the bicarbonate.
 - (2) Is the degree of respiratory compensation for metabolic acidosis—
 - (a) Too much ($\text{pH} \geq \text{normal}$) = superimposed respiratory alkalosis; common clinical settings include:
 - i. Salicylate poisoning
 - ii. Sepsis
 - iii. Early cyanide poisoning
 - iv. Increased intracranial pressure with shock or seizure
 - v. Normal renal compensation for primary respiratory alkalosis
 - (b) Too little ($\text{change in } p\text{CO}_2 < 1.5 \times \text{bicarbonate} + 8$) = superimposed respiratory acidosis; common clinical settings include:
 - i. Sedative-hypnotic overdose with shock or seizures
 - ii. Patient with primary ventilatory impairment and metabolic acidosis
 - iii. Partial renal compensation for primary respiratory acidosis
 - (3) Is the magnitude of the increase in the anion gap equal to the magnitude of the decrease in serum bicarbonate? If not, is it:
 - (a) Greater? If the bicarbonate is relatively increased, this indicates a superimposed metabolic alkalosis. Common clinical settings include:
 - i. Vomiting patient with diabetic ketoacidosis or alcoholic ketoacidosis
 - ii. Administration of NaH_2CO_3 to an acidemic patient
 - (b) Less? If the chloride is relatively increased, this indicates an associated hyperchloremic metabolic acidosis. Common clinical settings include:
 - i. Renal excretion of ketones with retention of chloride (especially during initial treatment of diabetic ketoacidosis)
 - ii. Resuscitation with chloride-containing solution and dilution of serum bicarbonate
 - iii. Severe diarrhea, renal tubular acidosis, and any primary cause of metabolic acidosis (eg, shock, sepsis)

B. Metabolic acidosis

1. Definition: $\downarrow \text{pH}$ (<7.35) and $\downarrow \text{HCO}_3^-$ ($<20 \text{ mEq/L}$)
2. One of the most common causes of metabolic acidosis in the emergency setting is lactic acidosis from increased anaerobic metabolism due to hypoxia, low perfusion states, or intense muscular activity (such as during seizures). Another cause of lactic acidosis is impairment of oxidative phosphorylation by toxins such as iron, salicylates, and cyanide.
3. Clinical presentation
 - a. Kussmaul respiration
 - b. Cardiac depression

- c. Catecholamine hyporesponsiveness
- d. Cardiovascular collapse
- 4. If you categorize the patient as having an anion or nonanion gap acidosis, the cause is easier to determine.
 - a. Normal anion gap acidosis (\uparrow Cl^-)
 - (1) Associated with hypokalemia
 - (a) GI losses
 - i. Diarrhea
 - ii. Ureteroenterostomy
 - (b) Renal losses
 - i. Renal tubular acidosis
 - ii. Acetazolamide
 - (2) Associated with hyperkalemia
 - (a) Early renal insufficiency
 - (b) Adrenal insufficiency
 - (c) Post-hypocapnia
 - b. Increased anion gap acidosis (normal Cl^-)
 - Alcoholic ketoacidosis
 - Methanol, Metformin
 - Uremia
 - Diabetic (or alcoholic) ketoacidosis
 - Paraldehyde, Phenformin
 - Isoniazid, Iron, Inhalant (carbon monoxide, hydrogen sulfide) poisoning (mitochondrial injury caused by reverse transcriptase inhibitors [HIV treatment])
 - Lactic acidosis \rightarrow (shock, hypoxia, seizures, cyanide, metformin, phenformin)
 - Ethylene glycol
 - Salicylates, Solvents
- 5. Management
 - a. Controversial
 - b. All agree that treatment of the underlying cause is paramount.
 - c. When the underlying cause is not readily reversed, some authors advocate administration of sodium bicarbonate to maintain a pH >7.10 or a serum bicarbonate >5 mEq/L.
 - d. Bicarbonate can precipitate rapid electrolyte disturbances, resulting in symptomatic hypokalemia and hypocalcemia.
 - e. Theoretically, paradoxical cerebral acidosis can occur because of the differential ability of CO_2 and HCO_3^- to cross the blood-brain barrier if bicarbonate is given too rapidly, ie, if CO_2 crosses readily, HCO_3^- crosses slowly.
 - f. Specific therapy of the underlying cause
 - (1) Insulin (diabetic ketoacidosis)
 - (2) Pressors, antibiotics, and fluids (lactic acidosis)
 - (3) Dialysis (renal failure, methanol, ethylene glycol, salicylates)
 - (4) Ethanol or 4-methylpyrazole (methanol, ethylene glycol); 4-methylpyrazole (an alcohol dehydrogenase antagonist) is an alternative substitute for IV ethanol.

- (5) Titrated HCO_3^- (renal tubular acidosis, according to type)
- (6) Benzodiazepines (status epilepticus)

C. Metabolic alkalosis

1. Definition: \uparrow pH (>7.45) and \uparrow HCO_3^- (>26 mEq/L)
2. Most common reasons for a primary increase in blood bicarbonate concentration
 - a. Vomiting and nasogastric suctioning
 - b. Diuretic administration
 - c. Adrenocortical hormone excess
3. Metabolic alkalosis can be categorized as "chloride sensitive" or "chloride resistant."
 - a. Chloride-sensitive alkalosis (saline responsive): common
 - (1) Diuretics \rightarrow loss of K^+ and Cl^- \rightarrow replace with saline and potassium
 - (2) Vomiting and nasogastric suctioning \rightarrow loss of H^+ , K^+ , and Cl^- \rightarrow (\uparrow renal excretion) \rightarrow replace with saline
 - b. Chloride-resistant alkalosis (saline unresponsive): rare
 - (1) Mineralocorticoid excess \rightarrow \uparrow renal absorption of Na^+ and HCO_3^- with \uparrow renal excretion of K^+ , H^+ , and Cl^- \rightarrow replace with potassium
 - (2) Large amounts of potassium are usually required to reduce renal excretion of H^+ and treat the underlying cause.

D. Respiratory acidosis

1. Definition: \downarrow pH (<7.40) and \uparrow pCO_2 (>45 mmHg)
2. Primary cause is inadequate ventilation and/or increased dead space, both of which are associated with \downarrow excretion of pulmonary CO_2
 - a. Etiology
 - (1) Head or chest trauma
 - (2) Oversedation
 - (3) Metabolic coma
 - (4) Neuromuscular disorders
 - (a) Neuropathies
 - (b) Myopathies
 - (5) Chronic hypoventilation in obese patients
 - (6) Obstruction
 - (a) Foreign body
 - (b) Bronchospasm or laryngospasm
 - b. Cause of increased dead space (with or without inadequate ventilation) \rightarrow COPD
3. Full renal compensation requires 48 hours of steady state alteration. Compensation is not expected to be complete; however, the pH may "normalize" in some patients.
4. Proceed with caution when treating the underlying disorder.
 - a. The pCO_2 should not be lowered more than 5 mmHg/hr (especially in those with chronic compensated respiratory acidosis, eg, patients with COPD).
 - b. Ventilator assistance: continuous positive-airway pressure (CPAP) or bi-level positive-airway pressure (BiPAP) may be necessary in selected patients.
 - c. Low-flow oxygen should be given to Pickwickian patients.

E. Respiratory alkalosis

1. Definition: \uparrow pH (>7.45) and \downarrow $p\text{CO}_2$ (<35 mmHg); CO_2 excretion exceeds CO_2 production.
2. Etiology
 - a. Anxiety with hyperventilation (most common clinical setting in the emergency department)
 - b. Primary CNS disorders
 - c. Hypermetabolic states
 - d. Conditions associated with hypoxia
 - e. Hepatic insufficiency
3. Renal compensation results in \downarrow serum bicarbonate.
4. Management
 - a. Aimed at the underlying cause
 - b. It is no longer recommended to have patients rebreathe expired air. Mild sedation may be useful in anxious patients.

F. Lactic acidosis

1. Etiology
 - a. Lactate is a normal product of anaerobic metabolism, so an increased serum lactate level does not necessarily mean that the patient has lactic acidosis. Several causes of hyperlactemia may not produce significant clinical consequences.
 - (1) Acute conditions (not usually associated with lactic acidosis)
 - (a) Exercise
 - (b) Hyperventilation
 - (c) Infusions of glucose, saline, or bicarbonate
 - (d) Injections of insulin or epinephrine
 - (2) Chronic conditions (sometimes associated with lactic acidosis)
 - (a) Severe congestive heart failure
 - (b) Pulmonary disease
 - (c) Liver disease
 - (d) Diabetes mellitus
 - (e) Generalized seizures
 - b. The diagnosis of lactic acidosis is made when the arterial plasma lactate level is increased (>4 – 5 mEq/L) and there is an associated high anion gap acidosis. Other causes of high anion gap acidosis must be excluded to confirm this diagnosis:
 - (1) Ingestions
 - (a) Alcohols (methanol, ethylene glycol)
 - (b) Salicylate
 - (c) Paraldehyde
 - (d) Isoniazid, iron
 - (e) Cyanide
 - (2) Ketoacidosis (alcoholic and diabetic)
 - (3) Uremia

(4) A mnemonic for causes of high anion gap acidosis is "A MUD PILES" and cyanide.

Alcoholic ketoacidosis

Methanol, Metformin

Uremia

Diabetic (or alcoholic) ketoacidosis

Paraldehyde, Phenformin

Isoniazid/Iron/Inhalant (carbon monoxide, hydrogen sulfide) poisoning
(mitochondrial injury caused by reverse transcriptase inhibitors [HIV treatment])

Lactic acidosis → shock, hypoxia, seizures, cyanide, metformin, phenformin

Ethylene glycol

Salicylates

2. Management

- a. The presence of clinically significant lactic acidosis indicates a serious underlying disorder that must be identified and corrected.
- b. IV sodium bicarbonate has traditionally been the mainstay of therapy, but it remains a controversial issue. In general, sodium bicarbonate should be given only when the pH is ≤ 7.1 . The approximate dose in milliequivalents can be calculated from the following formula:

$$\text{HCO}_3^- \text{ deficit} = 25 - \text{measured HCO}_3^- \times 0.5 (\text{body weight in kg})$$

- c. Most do not favor the use of insulin (or glucose and insulin infusions) in the treatment of lactic acidosis. The exception is diabetic ketoacidosis with concomitant lactic acidosis.
- d. Thiamine should be given to alcoholic patients with lactic acidosis, because it is needed in the oxidation of pyruvate.

G. Important points to remember in the management of acid-base disorders

1. An acid-base abnormality due to mixed disorders may exist in the presence of a normal pH or CO_2 .
2. If you suspect that respiratory acidosis is due to opioid use, administer naloxone early in the clinical evaluation.
3. If you suspect a toxicant as the cause of an acid-base abnormality, you need specific quantitative serum levels of ASA, methanol, ethylene glycol and, if isoniazid can be detected by your laboratory, a qualitative level of this drug, which may be helpful. (Broad, routine urine and serum toxicology screens are not helpful.) Consider general interventions such as decontamination measures and specific interventions such as dialysis in methanol toxicity early in the treatment process.
4. Consider isopropyl ingestion in ketotic patients with a normal glucose and no anion gap; these patients have an osmolar gap.
5. Pearls and pitfalls of anion-gap acidosis
 - a. Nitroprusside tests (urine reagent strips, reagent tablets) measure only acetoacetic acid (not β -hydroxybutyric acid), which means that patients with diabetic ketoacidosis or alcoholic ketoacidosis may register falsely low ketones or no ketones at all.
 - b. Patients who have ingested methanol (eg, "Sterno," windshield wiper fluid) may present 24–48 hours after ingestion with abdominal pain, an intoxicated appearance (but no breath odor of alcohol), and blurred vision with a hyperemic optic disc ("blind drunk"). All cases of methanol toxicity should be treated with IV ethanol (or 4-methylpyrazole) and most require dialysis.

- c. Patients who have ingested ethylene glycol (antifreeze) present within 12–24 hours with an intoxicated appearance (but no breath odor of alcohol). Hypocalcemia and calcium oxalate crystals in the urine may be seen, but their absence does not exclude the diagnosis.
 - (1) Calcium oxalate crystals can also be seen in nontoxic ingestions (eg, tea, cola, spinach, rhubarb).
 - (2) In addition to IV ethanol (or 4-methylpyrazole) and dialysis, these patients also need thiamine and pyridoxine. IV calcium may also be indicated for symptomatic hypocalcemia.
- d. Repeat laboratory values may be of help. Laboratories make mistakes, too!

H. Clinical situations and associated laboratory findings

- 1. Starvation/dehydration (decrease in total body water but not extracellular fluid volume), eg, the stroke patient left unattended at home for several days
 - a. Hypernatremia (excess water loss)
 - b. Hypokalemia (continuing urinary losses)
 - c. Increased serum osmolality (increased sodium, metabolic acids)
 - d. Mild metabolic acidosis (ketones produced)
 - e. Slightly increased anion gap
- 2. Diarrhea/chronic laxative abuse
 - a. Hyponatremia (fecal loss)
 - b. Hypokalemia (fecal loss)
 - c. Hypobicarbonatemia (fecal loss)
 - d. Hyperchloremia (compensation for HCO_3^- loss)
 - e. Hyperchloremic metabolic acidosis
- 3. Vomiting
 - a. Hypernatremia or hyponatremia or normal serum sodium
 - b. Hypokalemia (potassium shifts intracellularly secondary to alkalosis; excess renal loss)
 - c. HCO_3^- concentration >30 mEq/L
 - d. Hypochloremia (Cl^- loss)
 - e. Metabolic alkalosis (H^+ loss)

METABOLIC DISORDERS

I. HYPOGLYCEMIA

A. Definition

1. Although there is significant individual variation, signs and symptoms of severe hypoglycemia do not usually occur until blood glucose falls below 50 mg/dL.
2. Signs and symptoms
 - a. Diaphoresis
 - b. Tremulousness, nervousness
 - c. Tachycardia
 - d. Altered sensorium (confusion, agitation, unresponsiveness)
 - e. Focal neurologic signs
 - f. Seizures

B. Types

1. Postprandial hypoglycemia
 - a. Occurs within 6 hours of a glucose load
 - b. Etiology
 - (1) Alimentary hyperinsulinism (most common)
 - (2) Early manifestation of non-insulin-dependent diabetes
 - (3) Fructose intolerance
 - (4) Leucine sensitivity
 - (5) Cialactemia
 - (6) Idiopathic
2. Fasting hypoglycemia
 - a. Occurs 5–6 hours after a glucose load
 - b. Etiology
 - (1) Islet-cell tumor of the pancreas
 - (2) Extrapancreatic tumor
 - (3) Hypopituitarism
 - (4) Hepatic disease
 - (5) Chronic renal failure
 - (6) Starvation
 - (7) Myxedema
 - (8) Autoimmune disease
 - (9) Prolonged strenuous exercise
 - (10) Adrenocortical insufficiency
 - (11) Late pregnancy

3. Exogenous hypoglycemia
 - a. Drug-induced
 - (1) Insulin: most common cause of hypoglycemia in diabetic patients
 - (2) Sulfonylureas: Depending on the agent used, the hypoglycemic effect is potentiated by certain medications; these patients should be observed.
 - (3) Non-sulfonylurea secretagogues (repaglinide, nateglinide): less hypoglycemia than sulfonylureas
 - b. Alcohol (common cause)
 - (1) Primarily due to inhibition of gluconeogenesis (metabolism of alcohol → depletion of NAD → inhibition of gluconeogenesis)
 - (2) Depletion of liver glycogen stores (in the chronic, malnourished alcoholic patient) also plays a role.
 - c. Salicylate (hypoglycemic seizures in children are seen with overtreatment and overdose)
 - d. Propranolol and other β -blockers (may induce, potentiate, or mask signs and symptoms of hypoglycemia in diabetic patients)
 - e. Haloperidol
 - f. Phenothiazines
 - g. Disopyramide (usually occurs in malnourished, elderly patients)
 - h. Phenylbutazone
 - i. Monoamine oxidase inhibitors
 - j. Cimetidine
 - k. Pentamidine
 - l. Metformin (with concomitant alcohol use)
 - m. Sepsis
4. Hypoglycemia of infancy
 - a. Occurs in 4 out of 1,000 live births and is seen in:
 - (1) Infants of diabetic mothers
 - (2) Premature infants
 - (3) Infants that are small for gestational age
 - (4) Infants of narcotic-abusing mothers
 - b. Clinical presentation
 - (1) Seizures
 - (2) Limpness, lethargy
 - (3) Tremors
 - (4) Hypothermia
 - (5) Respiratory distress, apnea
 - (6) Color changes
5. Diagnostic evaluation: check blood glucose of critically ill child
6. Management
 - a. If the patient is awake and alert, he or she can ingest a meal of complex carbohydrates.
 - b. IV glucose is most effective.
 - c. IM glucagon may be used when IV access is not readily obtainable if the patient is not elderly or alcoholic (inadequate glycogen stores).

II. DIABETIC KETOACIDOSIS

A. Etiology

1. Actions of insulin
 - a. Augments glucose uptake by cells
 - b. Augments glucose uptake by the liver and its storage as glycogen
 - c. Increases lipogenesis and prevents lipolysis → increased levels of triglycerides
 - d. Inhibits gluconeogenesis and glycogenolysis → increases stored glucose as glycogen
2. Metabolic derangements are due to a relative insufficiency of insulin and an excess of the stress hormones (catecholamines, cortisol, glucagon, growth hormone, and somatostatin). Glucagon has been implicated as the primary hormone responsible for hyperglycemia and ketonemia in diabetic ketoacidosis.
 - a. Insulin insufficiency → inability of glucose to enter cells → hyperglycemia and cellular starvation
 - b. Cellular starvation → release of stress hormones → increased gluconeogenesis, glycogenolysis, and lipolysis → further hyperglycemia and increased free fatty acids
 - c. Free fatty acids → ketones (β -hydroxybutyrate and acetoacetate)
3. Precipitating factors
 - a. Lack of insulin
 - b. Infection (a main contributor to high mortality)
 - (1) Pneumonia
 - (2) Urinary tract infection
 - c. Acute myocardial infarction (a main contributor to high mortality)
 - d. Cerebrovascular accident
 - e. Trauma/surgery
 - f. Pregnancy
 - g. Alcohol/steroid use

B. Clinical presentation

1. \uparrow Glucose → \uparrow osmotic load → \downarrow intracellular water → \uparrow intravascular water → osmotic diuresis → \downarrow total body water. This results in:
 - a. Hypotension, tachycardia, and dehydration
 - b. Decreased serum electrolytes with depletion of:
 - (1) Sodium: the dilutional effect of hyperglycemia depresses the serum sodium still further (1.6 mEq/L for each 100 mg/dL increase of serum glucose).
 - (2) Potassium (total body)
 - (3) Phosphorus
 - (4) Magnesium
 - (5) Calcium
2. Insulin lack → hyperglycemia (glucose cannot enter cells) and cellular starvation → release of stress hormones (especially glucagon) → increased lipolysis → ketogenesis. This results in:
 - a. Acidosis with a decrease in sodium bicarbonate
 - b. A fruity breath odor (from acetone)

- c. Hyperventilation (Kussmaul respirations)
- d. Hyperkalemia: potassium is initially increased, because the acidosis causes it to shift out of the cells in exchange for the hydrogen ion. However, correction of the acidosis may be associated with a profound hypokalemia.
 - (1) As the acidosis improves, hydrogen ions will move out of the cells, allowing potassium to move back in and the serum potassium level to decrease.
 - (2) Administration of insulin and glucose also allows potassium to move back into the cells, which further decreases serum potassium.
 - (3) The average total body potassium deficit is $\sim 3\text{--}5$ mEq/kg.

C. Differential diagnosis of metabolic causes of coma in diabetic patients

1. Diabetic ketoacidosis
2. Hypoglycemia
3. Hyperglycemic hyperosmolar nonketotic coma
4. Alcoholic ketoacidosis
5. Lactic acidosis
6. Uremic acidosis

D. Diagnostic evaluation

1. Bedside testing (done immediately)
 - a. Glucose reagent strip can rapidly differentiate hypoglycemia from hyperglycemia.
 - b. Urine can be checked for:
 - (1) Glucose: indicating hyperglycemia
 - (2) Ketones with the nitroprusside test: a positive test is consistent with the diagnosis of diabetic ketoacidosis. However, this test primarily detects acetoacetate and will be falsely negative if most of the ketones are in the form of β -hydroxybutyrate or acetone.
2. Blood pH (arterial blood gases) and serum CO_2 content \rightarrow confirms acidosis (\downarrow pH and \downarrow bicarbonate)
3. CBC (baseline)
4. Glucose \rightarrow hyperosmolarity secondary to hyperglycemia is the single most important determinant of the patient's mental status (serum osmolality >320 mOsm/kg = altered mental status)
5. Serum ketones \rightarrow the following are usually increased:
 - a. Acetoacetate
 - b. β -hydroxybutyrate (which is not measured by the serum ketone level)
 - c. Acetone (which is chemically neutral)
6. Electrolytes \rightarrow in addition to identifying abnormalities, you need to determine the anion gap.
 - a. In the presence of a nondiagnostic ketone level, a wide gap suggests the presence of β -hydroxybutyrate as the major ketone.
 - b. In patients with mixed acid-base disturbances, the arterial pH may not accurately reflect the degree of acidosis; an anion gap does.
7. Calcium, magnesium, phosphorus \rightarrow if one is low, the other two generally are low as well.
8. BUN/creatinine (baseline)
9. Urinalysis, blood cultures, and chest radiograph \rightarrow look for infection. If the patient is septic, consider ultrasound to exclude emphysematous pyelonephritis.

10. ECG → look for evidence of acute myocardial infarction; estimate serum potassium level.
11. Laboratory summary for diabetic ketoacidosis
 - a. Blood glucose >250 mg/dL
 - b. Serum acetone >2:1 dilution
 - c. Bicarbonate <15 mEq/L
 - d. pH <7.30

E. Management

1. IV fluids in large volumes
 - a. Minimum requirements include at least 2 L in the first 2 hours (with the first liter being given in ≤30 minutes), followed by another 2 L over the next 4 hours, and another 2 L over the next 6 hours with careful monitoring of the serum glucose. As much as 5 L may be required during the first 3–4 hours.
 - b. If the patient is severely dehydrated, use normal saline for the initial 2 L of fluid resuscitation, then switch to 0.45% normal saline. In all other cases, alternating normal saline and 0.45% normal saline is recommended.
 - c. When the serum glucose level falls to ~250 mg/dL, add glucose to the IV to prevent iatrogenic hypoglycemia and cerebral edema.
2. Insulin administration
 - a. Large doses are not usually required to reverse diabetic ketoacidosis. In addition, complications (hypoglycemia, hypokalemia) are more likely to develop with large-dose insulin therapy.
 - b. Continuous IV infusion of low doses of insulin (5–10 units/hr in adults and 0.1 unit/kg/hr in children) is simple, safe, and effective. Some patients (1%–2%) do not respond to low-dose insulin therapy (infection is the usual underlying mechanism); if a patient does not respond within 1 hour (about a 50 mg/dL drop in blood glucose), double the infusion rate or administer a bolus.
 - c. Onset of action is almost immediate; a priming IV bolus is not required.
 - d. Insulin should initially be held if potassium level is <3.2 mEq/L until potassium replacement has occurred (American Diabetes Association).
3. Sodium bicarbonate
 - a. Use is clinically controversial
 - b. Indicated if the arterial pH is <7.0 and there is evidence of one of the following:
 - (1) Decreased myocardial contractility (cardiogenic shock)
 - (2) Respiratory depression (hypo- rather than hyperventilation)
 - (3) Renal failure (defective ability to resorb or generate bicarbonate)
 - c. Possible detrimental effects of bicarbonate administration
 - (1) Cerebral acidosis (paradoxical cerebrospinal fluid acidosis)
 - (2) Reduced ability of oxygen to dissociate (release) from RBCs → delays fall of serum ketones
 - (3) Hypokalemia (drives potassium intracellularly → increases potassium requirement)
 - (4) Hyperosmolarity (worsens dehydration)
4. Potassium replacement
 - a. Early replacement is standard.
 - b. If there is initial hypokalemia, add 40–60 mEq KCl to the first liter of fluid (one-half normal saline) and run over 1 hour.

- c. If the initial potassium level is normal, add 20–40 mEq KCl to the second liter of fluid (normal saline) and run at 500 mL/hr.
- d. Monitor serum potassium every 30–60 minutes until stable.
- e. If oliguria is present, renal function studies (BUN/creatinine) must be evaluated; if abnormal, potassium replacement must be decreased.
- 5. Phosphate replacement
 - a. Controversial: can lead to hyperphosphatemia with secondary hypomagnesemia and hypocalcemia, thus predisposing the patient to seizures
 - b. Phosphate should never be given during the initial management of diabetic ketoacidosis and is not indicated unless the serum PO_4 level falls below 1 mg/dL. A commercial potassium phosphate preparation ($\text{KH}_2\text{PO}_4 + \text{K}_2\text{HPO}_4$) can be used.
 - c. As patients undergo therapy for diabetic ketoacidosis, phosphate shifts from extracellular to intracellular compartments. Hypophosphatemia develops within 6–12 hours in most cases and is most severe 24–48 hours after the start of insulin therapy.
- 6. Monitoring
 - a. Blood levels of the following should be reassessed every 1–2 hours:
 - (1) Glucose
 - (2) Acetone (ketones may persist >36 hours)
 - (3) Potassium
 - (4) Phosphorus (after initiating insulin therapy)
 - (5) CO_2
 - (6) pH
 - (7) Chloride
 - b. Continuous cardiac monitoring is useful in detecting early changes in the serum potassium level.

F. Complications of diabetic ketoacidosis

- 1. Disease-related complications
 - a. Aspiration of gastric contents (unconscious patient)
 - b. Venous stasis that can lead to deep-vein thrombosis
 - c. Disseminated intravascular coagulation
 - d. Acute myocardial infarction
 - e. Rhabdomyolysis
- 2. Therapy-related complications
 - a. Hypoglycemia
 - b. Hypokalemia, hypophosphatemia
 - c. Paradoxical spinal fluid acidosis
 - d. Cerebral edema
 - e. Alkalosis (from too much bicarbonate replacement)
 - f. Congestive heart failure (from overhydration)
 - g. ARDS (from overhydration)

G. Mortality from diabetic ketoacidosis

- 1. Elderly: sepsis, cardiopulmonary complications
- 2. Children and young adults: cerebral edema (onset usually <24 hours)

III. HYPERGLYCEMIC HYPEROSMOLAR NONKETOTIC COMA

A. Etiology

1. The typical patient is elderly with mature-onset diabetes; the condition is stressed by one of the following precipitating events:
 - a. Infection (especially gram-negative sepsis or pneumonia)
 - b. Myocardial infarction
 - c. Cerebrovascular accident
 - d. GI bleeding
 - e. Pyelonephritis
 - f. Uremia
 - g. Subdural hematoma
 - h. Medications (eg, thiazide diuretics, diazoxide, phenytoin, steroids)
 - i. Cushing syndrome (and other endocrinopathies)
 - j. Trauma (including burns)
 - k. Hyperalimentation
 - l. Pulmonary embolus
 - m. Heat-related illness
 - n. Mesenteric ischemia
 - o. Rhabdomyolysis
 - p. Noncompliance with insulin therapy
 - q. New onset diabetes
2. Pathogenesis: mild hyperglycemia and stress \rightarrow \uparrow insulin resistance \rightarrow \uparrow insulin levels \rightarrow \uparrow gluconeogenesis and glycogenolysis \rightarrow \uparrow glucose without the development of ketosis \rightarrow \uparrow osmolality \rightarrow intracellular dehydration and osmotic diuresis with sodium depletion \rightarrow hyperglycemia and hyperosmolality and dehydration without acidosis
3. This condition develops slowly, sometimes over a period of weeks.

B. Clinical presentation

1. Signs and symptoms
 - a. Only 10% of these patients actually present in coma. The typical presentation is myriad vague complaints that include weakness, fatigue, dehydration, anorexia, and exacerbation of comorbid disease.
 - b. The most pronounced and consistent findings in these patients are neurologic.
 - (1) Most patients manifest some alteration in mental status, ranging from confusion to coma; the degree of obtundation present is directly correlated with the patient's serum osmolality.
 - (2) Focal neurologic signs (hemiparesis, hemisensory loss, focal seizures) are not uncommon and often confuse the diagnosis, leading people to believe the patient has had a stroke.
 - c. Signs of dehydration are also apparent (postural hypotension, reflex tachycardia, dry mucous membranes, shrunk tongue, absence of sweating).
2. Hyperglycemic hyperosmolar nonketotic coma should be suspect in a middle-aged or elderly patient who presents with an altered level of consciousness and is profoundly

dehydrated. A high blood glucose without ketonemia and without acidosis brings the clinical picture in focus.

3. Mortality rate is 8%–25% (was higher in the past [~20%–60%]).

C. Diagnostic evaluation/laboratory findings

1. Blood glucose >400 mg/dL (may be >1,000 mg/dL)
2. Serum osmolality >315 mOsm/kg

A serum osmolality should always be ordered, but results may not be readily available. Calculate osmolality using the following formula:

$$(2 \times \text{Na}) + \frac{\text{BUN}}{2.8} + \frac{\text{Glucose}}{18}$$

3. Negative serum ketones
4. Blood pH → no acidosis

D. Management

1. Fluid resuscitation
 - a. No agreement exists as to the type of initial fluid replacement, but most authors recommend the following three guidelines:
 - (1) If the patient is in shock, use isotonic saline (0.9% NaCl).
 - (2) If the patient is hypertensive or has significant hypernatremia (>155 mEq/L), use hypotonic saline (0.45% NaCl).
 - (3) When the blood glucose reaches ~250 mg/dL, add glucose to the IV.
 - b. The average fluid deficit in these patients is 8–12 L. One-half of the estimated water deficit must be replaced during the first 8 hours and the balance during the next 24 hours. Estimated water deficit in an elderly patient = weight in kg × 0.5 × 0.2
2. Insulin administration
 - a. Regular insulin given by continuous IV infusion is the customary method; dosage is 0.05–0.1 unit/kg/hr. If an IV bolus or IM injection is chosen instead, the dosage is the same (0.05–0.1 unit/kg). In many cases, no additional insulin is required after the initial dose.
 - b. No insulin should be given after the blood glucose reaches ~300 mg/dL.
 - c. Patients who are insulin-naïve may experience a rapid drop in blood glucose; in these cases, periodic serum glucose determinations are important.
3. Potassium replacement
 - a. KCl (10–20 mEq/hr) should be given during the acute phase of therapy (24–36 hours).
 - b. It should be started within 2 hours of IV fluid and insulin therapy, or as soon as adequate renal function has been confirmed.
 - c. Total body potassium depletion in nonketotic hyperosmolar coma is usually greater than that in diabetic ketoacidosis.
4. Magnesium replacement: MgSO₄ (1–2 g) should be given in the first 2 L of fluid if the serum magnesium is low.

IV. ALCOHOLIC KETOACIDOSIS

A. Clinical presentation

Heavy ethanol consumption (or binge drinking) and decreased or no food intake for several days \rightarrow \downarrow insulin and \uparrow glucagon and ethanol-induced inhibition of gluconeogenesis \rightarrow lypolysis \rightarrow \uparrow ketoacid production (acetoacetate and β -hydroxybutyrate) by the liver \rightarrow nausea, protracted vomiting, and abdominal pain that begins 24–72 hours before presentation and usually terminates with consumption of any food or liquid (including alcohol) \rightarrow tachypnea (ketoacidosis) and tachycardia (dehydration)

B. Diagnostic evaluation

1. A high anion gap acidosis is characteristic ($\text{Na}^+ - [\text{Cl}^- + \text{HCO}_3^-] = >16$) and is due primarily to high levels of β -hydroxybutyrate.
2. The nitroprusside test does not detect β -hydroxybutyrate at all; it may be negative or only weakly positive even in the presence of pronounced acidosis. No practical laboratory test is available that can detect β -hydroxybutyrate. A negative nitroprusside test, together with a very low serum bicarbonate level, suggests ketosis with a high level of β -hydroxybutyrate.
3. The blood glucose level is not high (usually <200 mg/dL), which differentiates alcoholic from diabetic ketoacidosis.
4. The blood alcohol level is generally low or not detectable.
5. Hypokalemia, hyponatremia, and hypophosphatemia may be present.

C. Management

1. Administration of IV fluids and glucose is the mainstay of therapy.
 - a. A solution containing both saline and glucose (D5/normal saline alternating with D5/0.45% normal saline) should be used, because recovery is faster when glucose-containing solutions are used.
 - b. Approximately 3–6 L of fluid are required over 24–48 hours.
2. Thiamine 50–100 mg IV should be given before the IV fluids are started, because glucose administration can precipitate Wernicke disease in the alcoholic patient.
3. Correction of hypokalemia is with 30 mEq KCl IV or 30 mEq potassium orally.
4. Insulin is not indicated and may be dangerous, because these patients usually have a near-normal or low blood glucose.
5. Sodium bicarbonate therapy is generally unnecessary and should be avoided unless:
 - a. The pH is <7.10 or
 - b. The patient is deteriorating clinically (rapid thready pulse and hypotension or inability to compensate by hyperventilation) despite adequate IV fluid therapy.
6. Recovery is assessed via the patient's clinical status and a rising pH; it is not determined by the nitroprusside test, which will paradoxically become strongly positive as clinical improvement occurs (reversal of acidosis causes β -hydroxybutyrate to be converted to acetoacetate).

V. THYROID STORM

A. Etiology

1. Thyroid storm is seen most often in patients with Grave disease (diffuse toxic goiter), which is the most common cause of hyperthyroidism. It is usually precipitated by a stressful event and must be suspected and treated based on clinical impression, because there are no specific pathognomonic findings or immediately available confirmatory laboratory tests ($\uparrow T_4$).
2. Precipitating factors
 - a. Grave disease
 - b. Infection (lung infection is the most common precipitating event)
 - c. Diabetic ketoacidosis, hyperglycemic hyperosmolar nonketotic coma, and insulin-induced hypoglycemia
 - d. Events that increase circulating levels of thyroid hormones in susceptible patients
 - (1) Premature withdrawal of antithyroid drugs
 - (2) Administration of radioactive iodide
 - (3) Use of iodinated contrast studies (eg, intravenous pyelogram)
 - (4) Poisoning with thyroid hormone
 - (5) Administration of a saturated solution of potassium iodide to patients with nontoxic goiters
 - (6) Vigorous palpation of the thyroid gland in thyrotoxic patients
 - e. Vascular events (cerebrovascular accident, pulmonary embolism, visceral infarction)
 - f. Trauma (including burns, surgery)
 - g. Emotional stress
 - h. Myocardial infarction
3. Probable pathogenesis: untreated or undertreated hyperthyroidism and a stressful event \rightarrow adrenergic hyperactivity \rightarrow thyroid storm

B. Clinical presentation

1. Clinical presentation: A patient with early thyrotoxicosis looks like a "psych case." CNS hyperactivity (anxiety, restlessness, manic behavior) and emotional lability dominate the clinical picture. A progressive hyperkinetic toxic state ensues and the patient may even appear to be acutely psychotic. If the patient is also diaphoretic, tachycardic, and febrile, thyroid storm must be excluded. A history of diarrhea and hyperdefecation, when present, heralds impending storm. Cardiovascular compromise becomes evident (congestive heart failure \rightarrow refractory pulmonary edema \rightarrow circulatory collapse). When combined with progressive CNS dysfunction (mental confusion \rightarrow obtundation \rightarrow coma), death may occur within 72 hours.
2. Clinical clues to the diagnosis
 - a. History of hyperthyroidism
 - b. Tachycardia (100–170 beats per minute) associated with a wide pulse pressure (40–100 mmHg)
 - c. Exophthalmus, stare, or lid lag (ocular signs of Grave disease)
 - d. Palpable goiter
 - e. Myopathy (usually involves the proximal muscles); most common in the elderly

3. Diagnostic criteria

- a. Temperature $>100^{\circ}\text{F}$ (37.8°C)
- b. Marked tachycardia out of proportion to the fever
- c. CNS symptoms (excitation early \rightarrow CNS depression later)
- d. Cardiovascular or GI signs and symptoms
 - (1) Dysrhythmias (especially atrial fibrillation)
 - (2) Premature ventricular contractions
 - (3) AV block
 - (4) Severe diarrhea
 - (5) Nausea/vomiting
 - (6) Crampy abdominal pain

C. Apathetic hyperthyroidism

- 1. A rare form of thyrotoxicosis that usually occurs in patients ≥ 70 years old and is often misdiagnosed because the usual hyperkinetic manifestations are absent
- 2. Clinical presentation
 - a. Lethargy, slowed mentation, and placid apathetic facies
 - b. Goiter (may be small and multinodular)
 - c. Usual ocular signs of Grave disease are absent, but blepharoptosis (drooping upper eye lid) is common.
 - d. Excessive weight loss (average = 40 lbs)
 - e. Proximal muscle weakness
 - f. "Masked thyrotoxicosis"
 - (1) The usual clinical presentation is atrial fibrillation with CHF.
 - (2) In apathetic patients, cardiovascular signs and symptoms tend to "mask" thyrotoxicosis.

D. Management

- 1. General supportive therapy
 - a. Supplemental oxygen (\uparrow oxygen consumption in storm)
 - b. IV fluids and electrolytes (replace insensible and GI losses)
 - c. Cooling blanket and acetaminophen to control fever
 - d. Digitalis and diuretics for atrial fibrillation and CHF (atrial fibrillation may be refractory)
 - e. IV glucocorticoids (equivalent to hydrocortisone 300 mg/day)
 - (1) Increase the survival rate, but the mechanism is unknown
 - (2) Dexamethasone has an advantage over other glucocorticoids in that it decreases the peripheral conversion of T_4 to T_3 ; the dosage is 2 mg IV every 6 hours.
- f. Adrenergic blockade
 - (1) Propranolol
 - (a) Not only decreases sympathetic hyperactivity but also blocks, in part, the peripheral conversion of T_4 to T_3
 - (b) 1 mg/min IV with cautious incremental increases of 1 mg every 10–15 minutes to a total of 10 mg

- (2) Alternative agents
 - (a) Esmolol 500 mcg/kg IV bolus, then 50–200 mcg/kg/min
 - (b) Guanethidine 30–40 mg orally every 6 hours
 - (c) Reserpine 2.5–5 mg IM every 4–6 hours
- 2. Specific therapy
 - a. Antithyroid drugs: propylthiouracil and methimazole
 - (1) Both drugs block the synthesis of thyroid hormone but do not affect the release of stored hormone.
 - (2) Propylthiouracil has two advantages over methimazole.
 - (a) In addition to blocking thyroid hormone synthesis, it also inhibits conversion of T_4 to T_3 .
 - (b) It produces a more rapid clinical response.
 - (3) Both drugs are administered either orally or via nasogastric tube.
 - (a) Propylthiouracil 600–1,000 mg initially, followed by 200–250 mg every 4–6 hours until thyrotoxicosis is under control
 - (b) Methimazole 90–120 mg initially, followed by 30–60 mg/day
 - b. Iodide administration
 - (1) Inhibits thyroidal release of stored hormone
 - (2) Saturated solution of iodine should be given 1 hour after the loading dose of an antithyroid drug to prevent utilization of the iodide by the thyroid in the synthesis of new hormone; the dosage is 5 drops orally or through a nasogastric tube every 6 hours.
 - (3) Alternative agents
 - (a) Sodium iodide 0.5–1 g IV every 12 hours
 - (b) Lugol solution 8–10 drops orally every 6 hours
 - (c) Lithium carbonate 800–1,200 mg/day orally
- 3. Drugs to avoid in treating thyroid storm
 - a. Aspirin: may ↑ T_3 and T_4 levels
 - b. Sedatives: interfere with CNS assessment during therapy
 - c. Atropine: may accelerate heart rate and also counteract the effect of propranolol

VI. MYXEDEMA (HYPOTHYROID) COMA

- A. An endocrinologic emergency with a 30%–40% mortality rate
- B. Pathophysiology of hypothyroidism
 - 1. The hypothalamus releases thyrotropin-releasing hormone → stimulates the anterior pituitary to release thyroid-stimulating hormone (TSH) → stimulates the thyroid gland to release T_3 and T_4
 - 2. Hypothyroidism occurs when the normal release of T_3 and T_4 does not occur. The result is progressive slowing of all bodily functions.
 - 3. Hypothyroidism may be primary (intrinsic failure of the thyroid gland to release T_3 and T_4) or secondary to disease or destruction of the hypothalamus or pituitary gland. Primary

hypothyroidism is far more common (95% of cases) than secondary hypothyroidism (5% of cases).

a. Etiologies of primary hypothyroidism (TSH level is high)

- (1) Prior treatment of Grave disease with radioactive iodine or subtotal thyroidectomy (most common cause)
- (2) Autoimmune thyroid disorders (second most common cause)
 - (a) Primary hypothyroidism secondary to autoimmune destruction of the thyroid → glandular atrophy → thyroid failure
 - (b) Hashimoto thyroiditis
 - i. The most common cause of goitrous hypothyroidism in areas with adequate iodine
 - ii. Pathophysiologic mechanism may be defective hormone synthesis.
- (3) Rare causes of primary hypothyroidism
 - (a) Iodine deficiency
 - (b) Antithyroid drugs (lithium, phenylbutazone)
 - (c) Spontaneous hypothyroidism from Grave disease
 - (d) Congenital thyroid abnormalities

b. Causes of secondary hypothyroidism (TSH level is normal or decreased)

- (1) Pituitary tumors
- (2) Postpartum hemorrhage → postpartum pituitary necrosis
- (3) Sarcoidosis
- (4) Dysfunction of the hypothalamus ("tertiary hypothyroidism")

C. Evolution of hypothyroidism to myxedema and myxedema coma

1. Myxedema is a rare and severe form of hypothyroidism seen in patients with undiagnosed or inadequately treated hypothyroidism. Like hypothyroidism, myxedema is most prevalent in older women and occurs most often in winter.
2. The single most important factor in the evolution of myxedema to myxedema coma appears to be a stressful event; CHF and pulmonary infection are the most common. Other precipitating stressful events include:
 - a. Drugs (phenothiazines, narcotics, anesthetics, β -blockers, sedatives, amiodarone, lithium, iodide)
 - b. Trauma or exposure to cold
 - c. Stroke
 - d. CHF
 - e. Infection

D. Clinical presentation

1. Hypothermia (80% of cases) without sweating or shivering is typical.
2. Respiratory failure is also common and is characterized by:
 - a. Hypoxia (\downarrow pO_2)
 - b. Hypoventilation (respirations)
 - c. Hypercapnia (\uparrow pCO_2), which may lead to CO_2 narcosis (a major cause of the altered sensorium)

3. Hyponatremia (which is due to secretion of inappropriate antidiuretic hormone [SIADH]) is a potentially grave finding; can lead to water intoxication □ cerebral edema
4. Cardiovascular abnormalities
 - a. Hypotension
 - b. Bradycardia (most common ECG abnormality), prolonged QT interval, low voltage secondary to pericardial effusion
 - c. Cardiomegaly (seen on chest radiograph)
5. Evidence of hypothyroidism may be minimal or absent.
 - a. A history of hypothyroidism should be sought in any comatose patient with hypothermia and respiratory failure. Historical clues:
 - (1) Previous thyroid medication, radioactive iodine therapy, or subtotal thyroidectomy
 - (2) Previous complaints of fatigue, weakness, cold intolerance, weight gain, menstrual irregularity, and muscle cramps
 - (3) Previous evidence of neuropsychiatric abnormalities
 - (a) Psychiatric disorders or personality changes
 - (b) Seizures
 - (c) Cerebellar signs (ataxia, intention tremors, nystagmus, difficulty with coordinated movements)
 - (4) Previous history of paresthesias; a mononeuropathy is typical, with median nerve involvement (carpal tunnel syndrome) being the most common.
 - b. Look for evidence of hypothyroidism on physical examination.
 - (1) Bradycardia — if present, order the following:
 - (a) ECG → low voltage and nonspecific ST and T wave changes (present in ~50% of patients with pericardial effusion)
 - (b) Chest radiograph → exclude cardiomegaly
 - (2) Thyroidectomy scar
 - (3) Thin eyebrows and scant body hair
 - (4) Dry, scaly, yellow skin and puffy eyes
 - (5) Abdominal distention due to fecal impaction, ascites, ileus, or urinary retention
 - (6) Pseudomyotonic reflexes (brisk upstroke, slow relaxation), especially at the ankle
 - (7) Nonpitting edema, including periorbital

E. Diagnostic evaluation

1. Although thyroid function tests (TSH, thyroxine-binding globulin, free T₄) should be ordered and can confirm the diagnosis of hypothyroidism, results are not always available in a timely fashion; initial therapy must be based on clinical impression.
2. Characteristic laboratory findings
 - a. Arterial blood gases: ↓ pO₂ and ↑ pCO₂
 - b. Electrolytes: ↓ Na⁺ and ↓ Cl⁻ (and ↓ Ca⁺⁺ in thyroidectomized patients)
 - c. Glucose: normal or low
 - d. CBC: a left shift in the differential (with or without an increased WBC count) may be present if infection is the precipitating event.
3. Nonspecific associated laboratory findings
 - a. ↑ serum cholesterol (66% of cases)

- b. ↑ creatine phosphokinase, lactate dehydrogenase, and aspartate aminotransferase (occasionally)
- c. ↑ cerebrospinal fluid protein >100 mg/dL (in most hypothyroid patients)

F. Management

1. Supportive measures
 - a. Oxygen and ventilatory support for respiratory failure
 - b. Slow rewarming of hypothermic patients
 - c. Correction of hyponatremia by fluid restriction (for dilutional hyponatremia) or hypertonic saline and furosemide (for severe hyponatremia)
 - d. Glucose infusion if there is hypoglycemia
 - e. Thyroid hormone and vasopressors for hypotension
 - f. Antibiotics for underlying infection (consider empiric therapy until culture results are available)
 - g. Hydrocortisone 300 mg/day to protect against adrenal insufficiency
2. Specific therapy with thyroid hormone
 - a. The most critical aspect of therapy because clinical improvement (even with other supportive measures) will not be fully effective until thyroid hormone is replaced.
 - b. The drug of choice is IV thyroxine (300–500 mcg infused slowly, followed by 50–100 mcg/day).
3. Identify and treat comorbid conditions (usually infection).

VII. ADRENAL INSUFFICIENCY (ADDISON DISEASE) AND CRISIS

A. Pathophysiology

1. Anatomy and physiology
 - a. Adrenal cortex produces glucocorticoids and mineralocorticoids (which are essential for life), as well as a small amount of androgens (which do not play a significant role in the pathogenesis of Addison disease).
 - (1) The major glucocorticoid is cortisol, which is produced by the following mechanism: hypothalamus releases corticotropin-releasing factor → pituitary secretes adrenocorticotropic hormone (ACTH) → adrenal cortex produces and secretes cortisol
 - (2) The major mineralocorticoid is aldosterone, which is regulated by the renin-angiotensin system and by plasma potassium concentrations.
 - b. Adrenal medulla → secretes catecholamines (epinephrine and norepinephrine); no definite clinical condition has been ascribed to hypofunction of the medulla.
2. Hypofunction of the adrenal cortex
 - a. When the physiologic demand for glucocorticoids and mineralocorticoids exceeds the capacity of the adrenal cortex to produce them, adrenal insufficiency occurs.
 - (1) Primary adrenal insufficiency (Addison disease) is due to disease/destruction of the adrenal cortex or adrenalectomy.

- (a) Idiopathic atrophy, usually autoimmune mediated, is the leading cause of chronic adrenal insufficiency (70%–75% autoimmune, and 25%–30% truly idiopathic). Associated diseases include:
 - i. Diabetes mellitus
 - ii. Hashimoto thyroiditis
 - iii. Grave disease
 - iv. Hypoparathyroidism
 - v. Pernicious anemia
 - vi. Primary ovarian failure
- (b) Infectious/infiltrative causes
 - i. Tuberculosis
 - ii. Protozoal/fungal
 - iii. Sarcoidosis/amyloidosis
 - iv. AIDS/cytomegalovirus/herpes simplex virus
 - v. Hemochromatosis
 - vi. Metastatic cancer
- (c) Pharmacologic causes
 - i. Methadone
 - ii. Rifampin
 - iii. Ketoconazole
- (d) Causes of bilateral adrenal hemorrhage (adrenal apoplexy) that can lead to insufficiency or crisis
 - i. Anticoagulant therapy (especially after myocardial infarction)
 - ii. Fulminant neonatal sepsis (especially meningococcemia, ie, Waterhouse-Friderichsen syndrome)
 - iii. Hemorrhage in the newborn
- (2) Secondary adrenal insufficiency due to dysfunction or destruction of the pituitary (and tertiary insufficiency due to hypothalamic dysfunction) leads to inability of the pituitary gland to secrete ACTH.
 - (a) The most common cause of tertiary insufficiency (and crisis) is iatrogenic adrenal suppression from prolonged steroid use.
 - (b) Causes of secondary insufficiency
 - i. Pituitary tumor, hemorrhage, or postpartum infarction
 - ii. Granulomatous/infiltrative disease (sarcoidosis, hemochromatosis, histiocytosis X)
 - iii. Head trauma (basilar skull fracture)
 - iv. Infection (meningitis, cavernous sinus thrombosis)
 - v. Internal carotid artery aneurysm
 - vi. Hemodynamically unstable patients who required prolonged, high-dose vasopressor therapy
 - b. When the adrenal reserve is exhausted (primarily of cortisol) in patients with chronic adrenal insufficiency who are subjected to stress or a concurrent illness, adrenal crisis occurs.

- (1) Most common cause is abrupt withdrawal of steroids in a patient whose adrenal function has been suppressed by prolonged steroid use.
- (2) Other causes
 - (a) Infections (especially those that lead to adrenal hemorrhage)
 - (b) Trauma, surgery, or burns
 - (c) Pregnancy
 - (d) Hyperthyroidism
 - (e) Drugs (especially hypnotics or general anesthetics)

B. Clinical presentation

1. Primary adrenal insufficiency (Addison disease)
 - a. Overall, the patient appears weak and lethargic and exhibits fatigue on exertion. Postural hypotension and syncope are common. Heart sounds may be soft or inaudible.
 - b. Signs and symptoms of cortisol deficiency
 - (1) Lethargy
 - (2) Anorexia, nausea, vomiting
 - (3) Hypoglycemia with fasting
 - (4) Inability to withstand even minor stress without shock
 - c. Signs of aldosterone deficiency
 - (1) ↓ Heart size with cardiac output → postural hypotension, syncope, and azotemia (due to renal blood flow)
 - (2) Dehydration with sodium depletion and hyperkalemia
 - d. Prominent GI symptoms
 - (1) Anorexia, nausea, vomiting, and occasionally diarrhea with weight loss
 - (2) Abdominal pain
 - e. Cutaneous manifestations: brownish pigmentation over exposed areas of the body and over friction or pressure points such as the elbows, fingers, knees, toes, and nipples
2. Adrenal crisis
 - a. **Classic clinical scenario: The patient appears very ill, profoundly weak, and possibly confused. Hypotension (especially postural) is typical. Circulatory collapse, when present, may be profound (feeble, rapid pulse, and soft heart sounds). Fever is common. GI signs and symptoms are almost always present: anorexia, nausea, vomiting, and abdominal pain. Delirium and seizures may occur.**
 - b. **Laboratory findings are variable.**
 - (1) **Serum sodium is decreased (usual) or may be normal.**
 - (2) **Serum potassium is normal or slightly increased.**
 - (3) **Hypoglycemia is characteristic and may be profound.**

C. Diagnostic evaluation

1. Laboratory findings
 - a. Electrolyte abnormalities
 - (1) Hyponatremia (88%)
 - (2) Hyperkalemia (64%)
 - (3) Hypercalcemia (6%–33%)

- b. Hypoglycemia
- c. Azotemia (\uparrow BUN/creatinine)
- 2. ECG changes
 - a. Signs of hyperkalemia
 - (1) Tall peaked T waves
 - (2) Prolonged PR and QT intervals
 - (3) Absent P waves
 - b. Nonspecific changes
 - (1) Low voltage
 - (2) Flat or inverted T waves (in the absence of \uparrow K⁺)
 - (3) Depressed ST segment

D. Management

- 1. Primary adrenal insufficiency (Addison disease)
 - a. Glucocorticoid replacement with cortisone acetate (25 mg) or prednisone (5 mg) in the morning, followed by a half dose in the afternoon simulates the normal diurnal variation of cortisol secretion.
 - b. Mineralocorticoid replacement with fludrocortisone acetate 0.05–0.2 mg/day is a generally accepted regimen.
 - (1) The dosage should be reduced if hypertension develops.
 - (2) Supplemental intake of dietary salt is also indicated.
 - c. Androgen replacement is indicated in women with signs of androgen deficiency (scant axillary and pubic hair). The drug of choice is fluoxymesterone 2–5 mg/day orally.
- 2. Adrenal crisis
 - a. IV fluids: D5/normal saline with the first liter given over 1 hour. An additional 2–3 L may be needed during the first 8 hours of therapy.
 - b. Glucocorticoid replacement
 - (1) The drug of choice is dexamethasone (4 mg IV), because it does not interfere with measurement of steroids and it is long-acting (12–24 hours).
 - (2) Alternative therapy with hydrocortisone sodium succinate is also acceptable; the dose is 100 mg IV push plus 100 mg added to the first IV.
 - c. Administration of a mineralocorticoid is unnecessary in the initial phase of therapy in adrenal crisis, because the large doses of hydrocortisone provide adequate mineralocorticoid effect.
 - d. Hypoglycemia should be treated immediately with 50–100 mL D50W.

ENDOCRINE, METABOLIC, AND NUTRITIONAL DISORDERS: PRACTICE CLINICAL SCENARIOS

Answers immediately follow the clinical practice scenarios.

Scenario A

Presentation: A 60-year-old man presents with altered mental status and a blood glucose of 1,100 mg/dL.

What is the diagnosis?

Scenario B

Presentation: A 45-year-old homeless person presents with an altered mental status.

Diagnostic evaluation: Laboratory studies show an increased anion gap (>16 mEq/dL) and an increased osmolar gap (>10 mOsm/dL).

What is the diagnosis?

Scenario C

Presentation: A 25-year-old injects heroin IV and becomes apneic.

What is expected on the laboratory studies?

ANSWERS TO PRACTICE CLINICAL SCENARIOS

Scenario A

Diagnosis: hyperosmolar nonketotic coma

Diagnostic evaluation: Laboratory studies show hyperglycemia, ketoacidosis, and hypokalemia.

Scenario B

Diagnosis: isopropyl alcohol poisoning

Diagnostic evaluation: Differential diagnoses include uremia, methanol poisoning, and ethylene glycol poisoning.

Scenario C

Diagnostic evaluation: Laboratory studies show respiratory acidosis, metabolic acidosis, and positive opiates on toxicologic screen.

ENVIRONMENTAL DISORDERS

Burns.....	813
Pathophysiology.....	813
Diagnostic Evaluation.....	813
Management.....	815
Inhalation Injuries.....	817
Chemical Injuries.....	818
Electrical Injuries.....	818
Lightning Injuries.....	821
Heat Disorders.....	823
Cold Disorders.....	827
Frostbite.....	827
Hypothermia.....	829
Bites and Stings.....	833
Human Bites.....	833
Dog Bites.....	835
Cat Bites.....	836
Venomous Snake Bites.....	837
Bee Stings, Ant Bites, and Other Insect Bites and Stings.....	838
Black Widow Spider.....	838
Brown Recluse Spider.....	839
Scorpion Stings.....	840
Jellyfish Stings.....	841
Altitude Disorders.....	841
High-Altitude Disorders.....	841
Acute Mountain Sickness.....	841
High-Altitude Pulmonary Edema (HAPE).....	842
High-Altitude Cerebral Edema (HACE).....	842
Dysbarism.....	842
Submersion Injury.....	845
Other Environmental Hazards.....	851
Radiation Injuries.....	851
Crush Syndrome.....	851
Volcanic Eruptions.....	852
Blast Injuries.....	852

ENVIRONMENTAL DISORDERS: SELF-ASSESSMENT QUESTIONS

1. You are evaluating an adult burn victim of ideal body weight and find that he has partial-thickness burns of his entire right arm, anterior trunk, and one-half of his right leg. Using the "rule of nines," you estimate the total body surface area burned to be:
 - (a) 27%
 - (b) 31.5%
 - (c) 36%
 - (d) 54%
2. The initial fluid of choice for the resuscitation of burn victims is:
 - (a) D5/0.45% normal saline
 - (b) D5/lactated Ringer's
 - (c) Lactated Ringer's
 - (d) Colloids
3. Which of the following tissues has the least resistance?
 - (a) Nerves, blood vessels
 - (b) Bone and tendons
 - (c) Fat
 - (d) Skin
4. Which of the following statements regarding electrical injuries is false?
 - (a) Alternating current generally produces worse effects than direct current of the same voltage.
 - (b) The dysrhythmia produced by direct current is usually ventricular fibrillation.
 - (c) The exit wound produced by direct current is discrete.
 - (d) Lightning behaves like direct current.
5. All of the following statements regarding acclimatization are accurate except:
 - (a) It is mediated by aldosterone.
 - (b) It is a process through which an individual adapts to a warmer, more humid environment.
 - (c) It results in a decreased concentration of sodium in the sweat and in the urine.
 - (d) It results in decreased sweat volume.

6. The mechanisms by which lightning produces injury include all of the following except:
 - (a) Side flash ("splash")
 - (b) Blunt trauma
 - (c) Ground current
 - (d) "Freezing"
7. The appropriate method for rewarming a frostbitten extremity is to rewarm it rapidly in circulating water heated to:
 - (a) 100.4°–104°F (38°–40°C)
 - (b) 104°–107.6°F (40°–42°C)
 - (c) 107.6°–111.2°F (42°–44°C)
 - (d) 111.2°–114.8°F (44°–46°C)
8. All of the following ECG changes may be seen in association with hypothermia except:
 - (a) Osborn waves
 - (b) Prominent P and U waves
 - (c) T wave inversion
 - (d) Prolonged PR, QRS, and QT intervals
9. The effect of hypothermia on the uncorrected arterial blood gas is to produce a falsely:
 - (a) Low pH, high pO₂, and pCO₂
 - (b) High pH, low pO₂, and pCO₂
 - (c) Low pH, pO₂, and pCO₂
 - (d) High pH, pO₂, and pCO₂
10. Cat-scratch disease is caused by:
 - (a) A gram-positive coccus
 - (b) A gram-negative bacillus
 - (c) A virus
 - (d) The causative organism is unknown.
11. A patient presents with the complaint of a cat bite to her hand. You choose an antibiotic to cover the most common pathogen associated with cat bites, which is:
 - (a) *Staphylococcus aureus*
 - (b) Anaerobes
 - (c) *Eikenella corrodens*
 - (d) *Pasteurella multocida*

12. What is the most venomous scorpion in North America?
- (a) Arizona bark scorpion (*Centruroides* genus)
 - (b) Arizona hairy scorpion, aka desert hairy scorpion (*Hadrurus arizonensis*)
 - (c) Stripe-tailed scorpion, aka "devil" scorpion (*Hoffmannius* genus)
 - (d) Fat-tailed scorpion (*Androctonus crassicauda*)
13. All of the following are appropriate antibiotic regimens for the treatment of a human bite wound except:
- (a) Dicloxacillin plus ampicillin or penicillin
 - (b) Augmentin
 - (c) Cefuroxime
 - (d) Clindamycin
14. The single leading cause of toxic death in the United States is:
- (a) Cyanide
 - (b) Hydrogen sulfide
 - (c) Carbon monoxide
 - (d) Phosgene gas
15. All of the following statements regarding "middle ear squeeze" are accurate except:
- (a) Symptoms (which include ear fullness and pain) develop on descent.
 - (b) Divers usually begin to develop symptoms at 16–20 feet.
 - (c) Symptoms result from failure to equalize the pressure between the middle ear and the water due to dysfunction of the eustachian tube.
 - (d) Failure to abort the dive (or to return to the surface and then try a slower more controlled descent) will produce worsening pain and rupture of the tympanic membrane.
16. All of the following statements regarding the treatment of black widow spider bites are accurate except:
- (a) The antivenin is equine-derived.
 - (b) The mortality rate of these bites is rare.
 - (c) Bites to the lower extremities or genitalia can produce pain simulating an "acute abdomen."
 - (d) Antivenin should be administered to all patients.
17. The organ system that is most resistant to the effects of ionizing radiation is:
- (a) GI system
 - (b) CNS system
 - (c) Hematopoietic system
 - (d) Reproductive system

18. The LD50 of whole body ionizing radiation is:
- (a) 250 rad
 - (b) 350 rad
 - (c) 650 rad
 - (d) 850 rad
19. Which of the following measures is not considered to be useful in the treatment of patients with heat stroke?
- (a) Removing the patient from the heat source and completely undressing him or her
 - (b) Applying atomized tepid water to the skin and fanning the patient
 - (c) Administering an antipyretic agent (aspirin, ibuprofen, acetaminophen)
 - (d) Controlling shivering with a benzodiazepine or chlorpromazine
20. When treating heat stroke, active core cooling measures should be abandoned when the patient's core temperature reaches:
- (a) 96.8°F (36°C)
 - (b) 98.6°F (37°C)
 - (c) 100.4°F (38°C)
 - (d) 102.2°F (39°C)
21. The antibiotic of choice for treating wound infections caused by *Pasteurella multocida* is:
- (a) Penicillin
 - (b) Cephalexin
 - (c) Erythromycin
 - (d) None of the above
22. Although all of the following burns may occur in association with a lightning strike, _____ burns are pathognomonic.
- (a) Linear
 - (b) Punctate
 - (c) Fern-like
 - (d) Thermal
23. A 38-year-old man presents 48 hours after being bitten by a neighbor's dog and is clearly septic. He had not sought prior medical attention, because the wound was relatively superficial and the dog was known to him and appeared healthy. Except for a splenectomy 6 years ago (due to trauma), his past medical history is unremarkable. Gram stain of the wound reveals a gram-negative bacillus. The most likely organism responsible is:
- (a) *Eikenella corrodens*
 - (b) *Pasteurella multocida*
 - (c) *Capnocytophaga canimorsus*
 - (d) *Rochalimaea henselae*

24. All of the following antibiotics would be effective in treating the patient in the dog bite question (above) except:
- (a) Penicillin
 - (b) A cephalosporin
 - (c) Erythromycin
 - (d) An aminoglycoside
25. Permanent injury to the cochleovestibular system may occur in patients with:
- (a) Barotitis media
 - (b) Barotitis interna
 - (c) "Sinus squeeze"
 - (d) Barotrauma of ascent
26. The most common presentation of a pulmonary air embolism is a:
- (a) Neurologic event
 - (b) Cardiovascular event
 - (c) Pulmonary event
 - (d) Psychological event
27. A patient with glucose-6-phosphate dehydrogenase deficiency has been bitten by a brown recluse spider. Which of the following medications is contraindicated?
- (a) Erythromycin
 - (b) Antihistamine
 - (c) Corticosteroids
 - (d) Dapsone
28. The best predictor of survival after radiation exposure is:
- (a) The whole body dose of ionizing radiation
 - (b) Timing of the onset of symptoms
 - (c) The absolute lymphocyte count 48 hours after exposure
 - (d) The organ system involved
29. On physical examination, the most sensitive indicator of a primary blast effect is:
- (a) Air emboli on fundoscopic examination
 - (b) Tympanic membrane rupture
 - (c) Petechial hemorrhages in the nasopharynx
 - (d) Tachypnea

30. Frequent metabolic derangements in submersion-injury victims include all of the following except:
- (a) Hypoxemia
 - (b) Acidosis
 - (c) Serum electrolyte abnormalities
 - (d) Hypercapnia
31. All of the following statements regarding submersion injury are accurate except:
- (a) Saltwater drowning is more common than freshwater drowning.
 - (b) Males are more commonly affected than females, regardless of age.
 - (c) Children <5 years old and teenagers are most commonly affected.
 - (d) The final common pathway in both "wet" and "dry" drownings is profound hypoxemia.
32. All of the following statements regarding submersion injury are accurate except:
- (a) Freshwater drownings are more common than saltwater drownings.
 - (b) 10%–15% of drownings are "dry" (no water enters the lungs).
 - (c) The initial management of the submersion-injury victim is determined by the type of drowning that occurred (wet, dry, saltwater, freshwater).
 - (d) Death in these victims is usually due to hypoxia.
33. All of the following are true about cold-water drowning except:
- (a) The arterial blood gas determination in a patient whose body temperature is 75.2°F (24°C) does not require temperature correction.
 - (b) Most victims of freshwater drowning have evidence of hemolysis as a manifestation of aspiration of large volumes of hypotonic fluid.
 - (c) A 10-year-old child in asystolic cardiac arrest with a body temperature of 71.6°F (22°C) after a 30-minute submersion requires emergent cardiopulmonary bypass.
 - (d) Very few victims of saltwater drowning have evidence of hemoconcentration as a manifestation of aspiration of hypertonic salt water.
34. The most important cause of morbidity and mortality in submersion injury is:
- (a) Hypothermia
 - (b) Metabolic acidosis
 - (c) Hemolysis
 - (d) Hypoxia

ANSWERS

- | | | | | |
|------|-------|-------|-------|-------|
| 1. c | 9. a | 17. b | 25. b | 33. d |
| 2. c | 10. b | 18. b | 26. a | 34. d |
| 3. a | 11. d | 19. c | 27. d | |
| 4. b | 12. a | 20. d | 28. c | |
| 5. d | 13. d | 21. a | 29. b | |
| 6. d | 14. c | 22. c | 30. c | |
| 7. b | 15. b | 23. c | 31. a | |
| 8. b | 16. d | 24. d | 32. c | |

Use the pre-chapter multiple choice question worksheet (page xx) to record and determine the percentage of correct answers for this chapter.

I. BURNS

A. Pathophysiology

1. Application of heat or chemicals causes "burning" of the skin and underlying tissues. Loss of the normal skin barrier allows fluids and electrolytes "out" and bacteria "in."
2. Pulmonary damage occurs from direct heat, especially with steam exposure, as well as from inhalation of products of combustion and particulate matter.

B. Diagnostic evaluation

1. Obtain the following information:
 - a. Was the victim burned in an enclosed or open space?
 - b. Were there any toxic products of combustion present at the burn site?
 - c. Does the patient have any respiratory symptoms?
2. Estimate the body surface area (BSA) burned (and chart it).
 - a. The "rule of nines" is used for adults and children >4 years old. It should not be used for children <4 years old, because it does not correct for the changes that occur in BSA with growth. These changes are most prominent in the head and legs; as children develop, they grow out of their disproportionately large heads and into their legs.

Table 33: Body Surface Area

	Adults	Children
Head and neck	9%	18%
Each arm	9%	9%
Each leg	18%	13%
Anterior trunk	18%	18%
Posterior trunk	18%	18%
Perineum	1%	2%

- b. The "rule of nines" is less accurate in obese and pregnant patients.
 - c. Because the patient population is becoming increasingly obese, the accuracy of the "rule of nines" has become controversial. Therefore, the Parkland formula with a given BSA may be more likely to be asked on the exam.
 - d. The Lund and Browder chart may be used for adults, but it is especially useful for infants and young children because it is age-adjusted, allowing for changes in BSA due to growth.
 - e. "Rule of palms": The surface of the palm is approximately 1% of BSA. This method of estimating burn size is readily available and most appropriate for burns with an irregular shape or distribution.
3. Estimate burn depth (and chart it). The initial depth of a burn may actually increase within the first 24–48 hours because of ongoing edema and dermal ischemia.
 - a. First degree (superficial)
 - (1) Skin is red and painful; no blister formation
 - (2) Minimal or no tissue destruction
 - (3) Most common cause: sunburn
 - (4) Wound healing: 7 days (no scarring)

- b. Second degree (partial thickness)
 - (1) Superficial
 - (a) Skin is red, wet, and painful with blister formation.
 - (b) Epidermal and superficial dermal injury
 - (c) Most common cause: hot liquid
 - (d) Wound healing: 14–21 days (some scarring)
 - (2) Deep
 - (a) Skin is white, with some erythematous areas, less blanching and moisture, tender
 - (b) Deep reticular dermal injury
 - (c) Causes: hot liquid or oil, steam, flame
 - (d) Wound healing: 3–6 weeks (some scarring)
 - c. Third degree (full thickness; dermal structures involved)
 - (1) The skin is charred, pearly white, "leathery," and insensitive.
 - (2) The entire skin layer and its nerve endings are destroyed and cannot grow back.
 - (3) Causes: hot oil, steam, flame, high-voltage electrical burns
 - (4) Wound healing: does not occur without surgical repair, skin grafting, or both (significant scarring)
 - d. Fourth degree (musculoskeletal injury)
 - (1) Extends below dermis to subcutaneous tissue (bone, muscle fascia)
 - (2) Tissue is necrotic.
 - (3) Causes: hot objects with high specific gravity (eg, molten metal), electrical burns
4. Estimate the full extent of the burn (and chart it).
- a. Minor burns
 - (1) Partial thickness <15% total BSA (<10% if younger than 10 or older than 50 years)
 - (2) Full thickness <2% total BSA
 - b. Moderate burns
 - (1) Partial thickness 15%–25% total BSA (10%–20% if younger than 10 or older than 50 years)
 - (2) Full thickness 2%–10% total BSA
 - c. Major burns
 - (1) Partial thickness >25% total BSA (>20% if younger than 10 or older than 50 years)
 - (2) Full thickness >10% total BSA
 - (3) Burns to the face, hands, major joints, feet, or perineum
 - (4) Burns to an immunocompromised host
 - (5) Burns and associated inhalation injury or major trauma
 - (6) Electrical burns (high voltage)
 - (7) Circumferential burns of the chest or extremities
 - (8) Caustic chemical burns
 - d. High-risk patients
 - (1) Younger than 10 or older than 50 years with minor to moderate burns
 - (2) Concurrent medical illness or immunocompromised
 - (3) Associated head injury, stroke, or psychiatric illness
 - (4) Prolonged exposure in a confined space

C. Management

1. Prehospital

- a. ABCs first, including stabilization with high-flow oxygen and IV fluids, followed by a head-to-toe survey looking for signs of trauma
- b. Remove the patient's clothing (to stop the burning process), and cover the burn wounds with a clean dry sheet or sterile dressings.
- c. Severe pain may be lessened with frequent boluses of fentanyl IV, with dosage based on weight by medical protocol.

2. Hospital (always use aseptic technique)

a. ABCs: major pitfall is inadequate airway management

- (1) Make sure that the patient is receiving adequate supplemental oxygen; all patients with moderate to major burns should receive 100% oxygen via nonrebreather mask; place on pulse oximeter, but remember that the pulse oximeter may be inaccurate in the face of carbon monoxide and some other chemical inhalation injuries.
- (2) Early intubation is indicated if there are any signs of respiratory distress, including stridor, bronchospasm, or subjective breathing difficulty, because these may make it difficult or impossible to intubate later.
 - (a) Unconscious or obtunded patients exposed to fire or smoke who were found in an enclosed space → consider possible cyanide poisoning (see page 735)
 - (b) Moderate to severe respiratory distress (including tachypnea, stridor, or labored breathing)
 - (c) Mild respiratory distress and signs of inhalation injury
 - i. Facial burns
 - ii. Sooty sputum
 - iii. Singed facial/nasal hairs
 - iv. Oropharyngeal erythema, swelling, carbon deposits
 - (d) Abnormal laboratory parameters
 - i. Hypoxia despite 100% oxygen
 - ii. High carboxyhemoglobin
 - iii. Low peak flow

b. Fluid resuscitation

- (1) Patients with burns >20% total BSA require fluid resuscitation; start two large-bore IV lines (above waist level) with lactated Ringer's (the fluid of choice for the first 24 hours after burn).
- (2) Learn one fluid resuscitation formula (eg, the Parkland formula), and use it. A general rule is to administer 2–4 mL/kg per % total BSA burned/24 hours.
 - (a) Give the first half of these fluids over the initial 8 hours (from time of burn).
 - (b) Give the second half of these fluids over the next 16 hours.
 - (c) Daily maintenance fluids should be administered in addition to the fluids above, particularly in infants and small children.
- (3) The above formula represents only an initial estimate of the fluid requirements and should be adjusted according to the patient's response (urinary output, heart rate, and mentation); administer fluids at a rate sufficient to maintain the pulse <110 beats per minute, urine output 0.5–1 mL/kg/hr in adults (>1 mL/kg/hr in children), and a clear sensorium.

- c. Patients with extensive burns also need a Foley catheter (to monitor fluid resuscitation and check urine for myoglobinuria) and a nasogastric tube (prophylaxis for ileus).
- d. Avoid excessive cooling; do not apply ice to wounds or cool compresses to extensive burns.
- e. Medications
 - (1) IV narcotics for pain relief
 - (2) Tetanus prophylaxis (the only medication given IM)
 - (3) Prophylactic antibiotics are indicated only in children with sore throats or those with known streptococcal infections (scalded skin syndrome).
- f. Baseline studies for patients with major burns
 - (1) Arterial blood gases with a carboxyhemoglobin level (persistent metabolic acidosis + \uparrow carboxyhemoglobin level \rightarrow possible cyanide toxicity)
 - (2) ECG
 - (3) Chest radiograph
 - (4) Hematology panel
 - (a) CBC with differential
 - (b) Prothrombin time/INR and partial thromboplastin time
 - (c) Type and screen
 - (5) Chemistries
 - (a) Glucose
 - (b) Electrolytes
 - (c) BUN/creatinine
 - (d) Creatine kinase
 - (6) Urine studies
 - (a) Urinalysis
 - (b) Myoglobin
 - (c) Human chorionic gonadotropin (hCG)
- g. Burn care
 - (1) Circumferential burns
 - (a) Assess the respiratory status in patients with a circumferentially burned thoracic cage; signs of respiratory insufficiency warrant consideration of escharotomy.
 - (b) Elevate circumferentially burned extremities, and monitor distal neurovascular status (sensation and pulses); loss of Doppler ultrasound signals mandates an escharotomy.
 - (2) Major burns: cover burns with dry sterile sheets, and contact nearest burn center regarding any further management.
 - (3) Minor burns
 - (a) Apply cool compresses.
 - (b) Irrigate burn with sterile saline and gently cleanse with a mild soap.
 - (c) Debride all loose tissue and broken blisters.
 - (d) Large intact blisters (particularly tense ones) or blisters over very mobile joints can also be debrided or aspirated (controversial).

- (e) Apply a topical antibacterial agent (eg, bacitracin), and cover the wound with sterile dressing. Polymixin B is no longer recommended because of higher rates of contact allergic reaction. Silver sulfadiazine has fallen out of favor; it should be avoided in pregnant women in the late third trimester, because it may cause kernicterus and depigment skin (and because of cost).

h. Disposition

- (1) Minor burns: discharge with pain medications and follow-up in 24 hours
 - (2) Moderate burns: require hospital admission; patients with minor burns who are at high-risk or unreliable may also be appropriate for admission.
 - (3) Major burns (see page 814): should be transferred to a burn center; patients with moderate burns who are high-risk or unreliable should also be considered for transfer.
- i. Contact the regional burn center early if transfer is contemplated, and tailor your initial treatment to their standard regimen. In preparation for transfer, a protocol should be followed that includes:**
- (1) Adequate resuscitation
 - (2) Physician-to-physician contact
 - (a) Do not delay initial airway management, fluid resuscitation, placement of catheters and lines, or diagnosis of associated life-threatening injuries.
 - (b) Aggressive fluid resuscitation is especially important to maintain blood pressure and urine output to prevent renal failure; however, pulmonary edema and hypoxia can result from fluid overload (especially in elderly patients with inhalation injury).
 - (3) A burn transfer checklist
 - (4) A copy of the patient's completed chart

D. Inhalation injuries

- 1. Respiratory burns
 - a. Intubate early, because subsequent edema may make this difficult or impossible later on.
 - b. If at all possible, refer these patients to a regional burn center.
- 2. Poisonous gas inhalation
 - a. Phosgene gas or $\text{N}_2\text{O}_2 \rightarrow$ immediate or delayed pulmonary edema
 - b. Hydrogen sulfide (H_2S) \rightarrow rapid death from respiratory failure
 - c. Carbon monoxide (CO)
 - (1) The single leading cause of toxic deaths in the United States
 - (2) A high index of suspicion is a must, because signs and symptoms are vague and multiple; any fire victim who was in an enclosed space should be evaluated for CO poisoning.
 - (3) Treat all symptomatic patients because carboxyhemoglobin levels do not always correlate with severity of symptoms.
 - (a) Minimum treatment is administration of 100% oxygen via a nonrebreather mask until the patient is asymptomatic and the carboxyhemoglobin level is $<5\%$ – 10% . Because of the greater susceptibility of the fetus to CO poisoning, however, oxygen therapy must be continued five times longer in pregnant women to adequately treat the fetus.

- (b) Hyperbaric oxygen therapy (although controversial) is still used for:
 - i. Persistent symptoms after 4 hours of 100% oxygen therapy
 - ii. Carboxyhemoglobin level >25%–40% at any time (level used varies with hospital policy and chamber accessibility)
 - iii. Coma (or a history of ↓ level of consciousness)
 - iv. Pregnant patients who are symptomatic or have a carboxyhemoglobin level >15
 - v. Neonates
 - vi. Severe metabolic acidosis (pH <7.2)
 - vii. Cardiovascular dysfunction (including ischemia on ECG)

E. Chemical injuries

1. Burns cause damage by protein denaturation.
2. The offending chemical should be removed or diluted as quickly as possible (preferably at the scene), because tissue destruction continues until the causative agent is removed or inactivated.
3. Most chemicals should be irrigated with copious amounts of water or saline for 15–30 minutes; however:
 - a. Alkalis penetrate more deeply (due to liquefaction necrosis) and require longer irrigation; acids penetrate less deeply (due to coagulation necrosis).
 - b. Dry chemical particles (eg, lime) should be removed before irrigation.
 - c. Sodium, lithium, and potassium metals should be covered with mineral oil or excised. Large amounts of water, to dilute exothermic reaction, may be used if oil is not available.
4. Further specific treatment, eg, application of calcium gluconate to hydrofluoric acid burns, may then be instituted as indicated.

F. Electrical injuries

1. Pathophysiology

- a. The tendency of a tissue to resist flow of electrical current is called its resistance. Nerves, blood vessels, mucous membranes, and muscles have the least resistance (because they have a higher concentration of electrolytes) and are, therefore, good conductors; current flows easily (and in high volume) through these tissues, producing much damage. Skin, when dry, is intermediate in resistance but, when moist or wet, resistance decreases. Bone, tendon, and fat have the highest resistance.
- b. Life-threatening complications
 - (1) Cardiac arrest from ventricular fibrillation or asystole (most common cause of death in the acute phase of electric injuries is ventricular fibrillation)
 - (2) Respiratory arrest
 - (3) Cardiac dysrhythmias
 - (4) Complications due to massive muscle breakdown
 - (a) Myoglobinuric renal failure with secondary hyperkalemia
 - (b) Dysrhythmias resulting from electrolyte imbalance (↑ K⁺ and Ca⁺⁺)
 - (c) Progressive intravascular thrombosis may contribute to continued muscle and tissue damage.
- c. The most common cause of tissue hypoxia in an electrical fire (or any other fire) is carbon monoxide poisoning.

2. Types of electrical current
 - a. Alternating current (household and commercial)
 - (1) Produces explosive exit wounds.
 - (2) Effects are usually worse with AC than with DC current at the same voltage. Tetanic contractions of the flexor muscles, which are usually stronger than the extensors, cause the victim to "lock up" to the charge, prolonging the exposure and increasing the severity of the injury.
 - (3) Ventricular fibrillation occurs more commonly from AC current.
 - b. Direct current (industrial, batteries, welding supplies)
 - (1) Produces discrete exit wounds
 - (2) Asystole occurs more commonly from DC current.
3. Magnitude of current
 - a. Injuries are classified as either high voltage (>1,000 volts) or low voltage (<1,000 volts).
 - b. Generally speaking, the greater the voltage, the more severe the injury; thus, it is important to try to determine the voltage involved in any given accident.
4. Current pathway
 - a. Electrical current usually enters through the hand or skull and exits through the heel.
 - b. The path taken by the current determines tissues and organs at risk.
 - (1) Current passing through the head and neck is more likely to induce cataract formation than current passing distal to these sites.
 - (2) Current traversing the heart and thorax is associated with an increased likelihood of cardiac complications.
5. Clinical presentation and physical examination
 - a. Perform a head-to-toe survey looking for evidence of trauma.
 - b. Look for entrance and exit wounds.
 - (1) Electrical wounds are "bull's-eye" in appearance with a charred center; a grayish white area of coagulation necrosis is adjacent to the surrounding red, edematous tissue.
 - (2) The true extent of underlying injury will not be apparent initially, so do not debride the wounds in the emergency department. Electrical injury may cause progressive intravascular thrombosis.
6. Management
 - a. Initial assessment and stabilization
 - (1) ABCs
 - (2) Monitor, IV access, and oxygen
 - (a) Establish an IV line with a large-bore catheter; use lactated Ringer's or normal saline, and determine the rate based on the initial shock assessment (not the extent or degree of burn because most of the injury is internal); adjust the rate as needed to maintain a urine output of at least 0.5–1 mL/kg/hr.
 - (b) Give oxygen to everyone (no exceptions).
 - (c) Put the patient on a cardiac monitor.
 - (3) Foley catheter (monitor urine output)
 - (4) Nasogastric tube if injury is severe (increased risk of adynamic ileus)

- b. Diagnostic evaluation
 - (1) ABGs
 - (2) ECG
 - (3) Creatine kinase (CK) + CK-MB
 - (4) CBC
 - (5) Prothrombin time/INR and partial thromboplastin time
 - (6) BUN/creatinine
 - (7) Urinalysis (no RBCs but positive dipstick → presume myoglobinuria)
 - (8) Serum electrolytes (including Ca^{++})
 - (9) Urine myoglobin
 - (10) Radiographic studies as indicated
- c. Treat myoglobinuria.
 - (1) Administer fluids at a rate sufficient to maintain a urine output of 1.5–2 mL/kg/hr.
 - (2) If this does not clear the pigment, administer mannitol as a bolus of 25 g, and add 12.5 g to each subsequent liter of fluids (at a rate of 1 L/hr) until the myoglobin is cleared. Furosemide (2 mg/kg IV) may also be administered to promote diuresis.
 - (3) Alkalinization of the urine is suggested in some texts to be beneficial at CK concentrations >6,000 IU/L; however, this has never been proved.
- d. Do not forget tetanus prophylaxis.
- e. Disposition
 - (1) Hospitalization and cardiac monitoring are indicated for all patients with high-voltage burns and for those patients with low-voltage burns who are symptomatic (eg, chest pain, abnormal physical or laboratory findings). Those with severe electrical burns should be admitted to a burn center.
 - (2) Asymptomatic patients with low-voltage injuries may be discharged to home after a period of observation and cardiac monitoring in the emergency department if their physical examination and diagnostic evaluation are unremarkable (no skin lesions, normal ECG, no urinary heme pigment).
- f. Delayed complications of major electrical burns
 - (1) Myoglobinuric renal failure
 - (2) Compartment syndromes
 - (3) Gangrene (due to intravascular thrombosis)
 - (4) Dysrhythmias
 - (5) Infection
 - (6) Cataracts
 - (7) Neurologic problems (peripheral nerve damage, trouble with memory/concentration, seizures, delayed spinal cord syndromes)
- 7. Electrical lip burn
 - a. Clinical presentation: usual history is a child bites on an electrical cord, and burns lips and commissures.
 - b. Management
 - (1) Do not debride these wounds. Cleanse and apply a petroleum-based antibiotic ointment.
 - (2) These burns require close observation and referral to a plastic or oral surgeon for splinting and further care.

- c. **Complications:** Delayed hemorrhage from the labial artery may occur in 10%–15% of these patients 3–14 days after injury, when the eschar separates.

G. Lightning injuries

1. Pathophysiology

- a. Lightning injuries are extremely high-intensity bursts of direct current (10 million to 2 billion volts) that are very brief (0.1–1 milliseconds). Although one might compare lightning injuries to a massive DC countershock, the injuries that occur from a lightning strike are different from those that occur with man-made electricity, as is the treatment; unlike victims of high-voltage electricity, lightning victims rarely have deep burns or underlying tissue damage, and they do not generally require fluid resuscitation.
- b. Because of the brief duration, lightning rarely breaks down the skin; instead, it passes over the body surface, and if the victim's body is wet or sweaty, can cause burns in one of two ways:
 - (1) Moisture can be vaporized and produce "steam burns."
 - (2) Sudden expansion of water converting to steam can cause the clothes to "explode" off the body, burning it in the process.

2. Mechanisms of injury

- a. Direct strike: Lightning hits the victim directly and passes over or through him or her; morbidity and mortality are highest with this mechanism.
 - b. Contact: lightning strikes an object that the victim is touching.
 - c. Side flash or splash: lightning jumps from its initial strike site to a nearby person or object in its pathway.
 - d. Ground current or step voltage: lightning strikes the ground and spreads through it to the victim; if one of the victim's legs is nearer to the strike point than the other, a potential difference is created between them, resulting in the current passing through the legs and into the trunk.
 - e. Thermal burns occur as a result of boiling sweat, burning clothes, or hot jewelry.
 - f. Blunt trauma: lightning rapidly heating air followed by rapid cooling causes a blast effect that throws the victim; the organ affected most often by blast injury is the ear.
3. Classic clinical scenario: An observed strike is obvious, but many are not observed. An important diagnostic clue in the unconscious patient is that clothes may be partially missing, especially the shoes.
- a. Clinical picture (minor lightning strike): patient appears "stunned," is somewhat confused, and has difficulty recalling events that transpired a few hours ago. He or she has no memory of the event described. Unless the lightning strike was witnessed, this diagnosis may be missed. Do not be fooled by the absence of entrance or exit wounds; they are rare. The patient has mild hypertension and tachycardia and is likely to complain of headache. There is frequently a history of temporary loss of vision or hearing.
 - b. Possible physical findings (major lightning injuries)
 - (1) Cardiopulmonary injuries
 - (a) Cardiac arrest (asystole): most common cause of death
 - (b) Respiratory arrest secondary to tetany of diaphragm muscles: may be more prolonged than cardiac arrest and can cause secondary ventricular fibrillation
 - (c) Pulmonary edema
 - (d) Transient hypertension

- (2) Neurologic injuries
 - (a) Transient loss of consciousness (the most common neurologic event)
 - (b) Confusion and amnesia (both anterograde and retrograde) are also common.
 - (c) Peripheral nerve damage is another common finding.
 - (d) Seizures result from hypoxia or concomitant head injury.
 - (e) Transient flaccid paralysis (keraunoparalysis) of the lower extremities (which may initially appear cold, pale, pulseless, and insensitive) is generally due to vascular spasm and sympathetic instability; it usually resolves spontaneously over several hours.
- (3) Eye and ear injuries
 - (a) Immediate or delayed cataract formation
 - (b) Corneal injuries, uveitis, hyphema, vitreous hemorrhage, retinal detachment, optic nerve atrophy
 - (c) Unreactive dilated pupils secondary to autonomic instability (do not misinterpret as an indication of brain death)
 - (d) Ruptured tympanic membranes (>50% of cases) are caused by:
 - i. The blast effect or
 - ii. Basilar skull fractures or
 - iii. Direct burn
- (4) Skeletal injuries (fractures)
 - (a) Much less common than with manmade high-voltage injuries.
 - (b) Common sites are skull, spine, or long bones.
- (5) Effects on fetus
 - (a) Intrauterine death (25%)
 - (b) Neonatal death (25%)
 - (c) Healthy babies (50%)
- (6) Burns (four types)
 - (a) Linear: superficial burns that occur in areas where sweat and moisture accumulate.
 - (b) Punctate: small, discrete, circular burns that occur in clusters
 - (c) Lichtenberg figures: superficial fern-like burns that are pathognomonic for lightning strike
 - (d) Thermal: result from the ignition of clothing or the heating up of objects worn by the victim
- 4. Management
 - a. **"Resuscitate the dead" (even if there are multiple victims)**
 - (1) Those who are in cardiac or pulmonary arrest need immediate attention if they are to have any chance of survival.
 - (2) Those who are moving are OK on their own initially.
 - b. ABC assessment and initial stabilization followed by a head-to-toe survey for signs of trauma. Standard ACLS and ATLS treatment protocols should be followed. Massive fluid resuscitation is seldom indicated, because lightning injuries rarely produce significant tissue destruction.

- c. Burns, if present, should be treated in the usual manner. Do not forget tetanus prophylaxis.
- d. Baseline laboratory and diagnostic evaluation
 - (1) CBC
 - (2) Electrolytes, BUN/creatinine
 - (3) ECG and cardiac enzymes
 - (4) Urinalysis and urine for myoglobin
 - (5) Chest radiograph and other radiographs as indicated
 - (6) Brain CT (and cervical spine radiographs) should be ordered for patients with an altered level of consciousness.
- 5. Disposition: In almost all instances, these patients should be admitted with continuous cardiac monitoring for 24 hours.
- 6. Late sequelae
 - a. Posttraumatic stress disorder/keratunophobia (an abnormal fear of thunder and lightning)
 - b. Paresis
 - c. Impaired mental function
 - d. Memory deficits
 - e. Insomnia
 - f. Cataracts
 - g. Peripheral neuropathy

II. HEAT DISORDERS

A. Normal body temperature regulation (heat gain = heat loss)

- 1. Heat gain is the result of:
 - a. Metabolic activity
 - b. Environmental heat
 - c. Strenuous exertion
- 2. Heat loss results from:
 - a. Radiation
 - (1) Cutaneous vasodilation increases the amount of heat that can be dissipated through the skin.
 - (2) As ambient temperature approaches and surpasses body temperature, however, heat loss via radiation ceases and radiation becomes a source of heat gain.
 - b. Convection: heat loss due to air or water circulating across the body (wind chill)
 - c. Conduction: direct physical contact with a cooler object or immersion in water
 - d. Evaporation
 - (1) The degree of sweating is controlled by cholinergic and sympathetic activity.
 - (2) The effectiveness of sweating is decreased in the presence of high humidity.
- 3. The wet-bulb globe thermometer index
 - a. Accounts for the effects of humidity and radiant heat on the ambient temperature
 - b. The most accurate measure of environmental heat stress and the risk of heat illness

4. Acclimatization

- a. A process over a period of 1–2 weeks in which an individual adapts to a warmer, more humid climate via hypothalamic and thyroid adaptation
- b. Mediated by aldosterone, which reduces the sodium concentration in sweat and urine; this preserves fluid volume, thus preventing the development of a heat illness.
- c. Characterized by an earlier onset of sweating, greater sweat volume, and a decreased concentration of sodium in the sweat and in the urine

B. Heat illness

1. Imbalance between heat production and loss

2. Predisposing factors

- a. Physical activity (especially in a hot, humid environment)
- b. Extremes of age, poor physical condition, fatigue
- c. Inadequate indoor cooling (lack of or malfunctioning air conditioning)
- d. Excessive clothing
- e. Dehydration
- f. Preexisting cardiovascular disease
- g. Skin disorders
- h. Obesity

i. Drugs

(1) Phenothiazines (act centrally on the hypothalamus)

(2) Anticholinergics: reduce the ability to sweat

(a) Atropine

(b) Scopolamine

(c) Cogentin

(d) Antihistamines

(e) Cyclic antidepressants

(3) β -blockers/calcium channel blockers (\downarrow heat loss)

(4) Diuretics (\downarrow heat loss)

(5) Amphetamines (\uparrow heat gain) and sympathomimetics, including OTC pseudoephedrine and bath salts

(6) LSD (\uparrow heat gain)

(7) Cocaine (\uparrow heat gain)

(8) Monoamine oxidase inhibitors (\uparrow heat gain)

(9) MDMA/ecstasy, "rave drugs"

(10) Haloperidol (neuroleptic)

(11) Alcohol withdrawal

3. Pathophysiology

- a. Heat cramps: inadequate replacement of salt from loss through sweating \rightarrow hyponatremia \rightarrow muscle cramps
- b. Heat tetany: hyperventilation \rightarrow respiratory alkalosis \rightarrow paresthesias and carpopedal spasm
- c. Heat exhaustion: salt water depletion from sweat loss \rightarrow hypovolemia and hypoperfusion, and normal mental status and neurologic examinations

d. Heat stroke

- (1) Heat stress (classic) or endogenous heat production (exertional) → a breakdown of central thermoregulatory control → hyperpyrexia and neurologic symptoms ± absence of sweating. Sweating is usually absent in classic heat stroke and present in exercise-induced or exertional heat stroke. Cerebral edema is common.
- (2) Loss of sweating ability aggravates (but does not cause) the problem.
 - (a) The exact mechanism by which the ability to sweat is lost is unknown, but a direct thermal effect on sweat glands is a contributing factor.
 - (b) Anticholinergic drugs are the most frequent cause of impaired sweating in classic heat stroke.
 - (c) Specific disorders such as congenital anhidrosis, cystic fibrosis, and quadriplegia are rare contributing factors.
- (3) Volume depletion and electrolyte imbalance are not usually prominent features, but central venous pressure is usually increased.
- (4) Patients with preexisting cardiovascular disease have a reduced capacity to cope with the hemodynamic changes of peripheral vasodilatation.
- (5) End-organ and systemic injury can and does occur.
 - (a) Cardiac (CHF, pulmonary edema)
 - (b) ARDS
 - (c) Liver (marked increases of AST and ALT)
 - (d) Kidney (hematuria, proteinuria, acute tubular necrosis)
 - (e) Muscular (mild increase in creatine kinase → rhabdomyolysis and acute renal failure)
 - (f) Hematologic (altered coagulation → bleeding, disseminated intravascular coagulation)

C. Classic presentation

1. Heat cramps: The patient complains of severe muscle cramping typically involving the calves, thighs, and shoulders. Questioning reveals that the cramps developed after a bout of intense physical activity and profuse sweating, during which the patient had been replacing fluid losses with a hypotonic solution. Body temperature is normal.
2. Heat tetany: The patient is hyperventilating and complains of tingling and spasms of the hands and feet.
3. Heat exhaustion: The patient presents with extreme fatigue and profuse sweating. If the onset was fairly recent, he or she will complain of light-headedness, nausea, vomiting, and a dull headache. If the patient started feeling ill several hours ago, concerned family or friends may have brought the patient in because he or she "didn't look right;" this patient is tachypneic, tachycardic, and may be hypotensive. Body temperature in patients with heat exhaustion is normal or slightly increased.
4. Heat stroke: a true medical emergency characterized by an altered level of consciousness and any neurologic finding and an increased temperature
 - a. Classic heat stroke occurs most often in middle-aged or elderly patients who live a sedentary lifestyle and are taking medications for chronic illnesses. It is caused by environmental heat exposure. Do not be fooled if the patient is sweating; some do initially and, if not treated, will stop sweating and develop hot, dry skin. It may be difficult to exclude a cerebrovascular or CNS infection on physical examination. An accurate history is critical to the diagnosis; ataxia may be the initial (or presenting) complaint. Laboratory findings include:

- (1) Respiratory alkalosis and mild metabolic acidosis
- (2) Mild coagulopathy and increased creatine kinase
- (3) Normal glucose and calcium levels
- b. Exertional heat stroke typically occurs in young, healthy patients who have been engaged in strenuous exercise. These patients are usually diaphoretic on presentation. Characteristic laboratory findings include:
 - (1) Respiratory alkalosis and marked lactic acidosis
 - (2) Disseminated intravascular coagulation and rhabdomyolysis (urine specimen looks like "machine oil")
 - (3) ↑ BUN/creatinine (acute renal failure)
 - (4) Hypoglycemia and hypocalcemia

D. Management

- 1. Heat cramps
 - a. Oral or IV fluid and electrolyte replacement
 - b. Rest in a cool environment with a gradual return to normal environment and activity
- 2. Heat tetany
 - a. Removal from heat
 - b. Rebreathe expired air (bag-breathing)
 - c. Exclude electrolyte abnormalities
- 3. Heat exhaustion
 - a. Bedrest in a cool environment
 - b. Rapid IV fluid/electrolyte volume replacement, initially with normal saline
 - c. Baseline studies: CBC, glucose, electrolytes, BUN/creatinine, liver profile, and urinalysis
- 4. Heat stroke
 - a. Management
 - (1) Immediate, aggressive, rapid cooling down to 102.2°F (39°C). Morbidity is directly related to severity and duration of hyperthermia.
 - (a) Remove patient from heat source, and undress him or her completely.
 - (b) Apply atomized tepid water (not ice) to the skin, and fan the patient.
 - (c) Apply ice packs to axillae and groin.
 - (d) Iced gastric lavage is rarely used; if done, monitor ins and outs.
 - (e) Immersion in ice water is also very effective but may precipitate seizures; monitoring and resuscitation are difficult with this technique. It is recommended, however, if above measures have failed to lower body temperature to 102.8°F (38.9°C) within 30 minutes.
 - (2) Supportive measures
 - (a) Administer high-flow oxygen to all patients, and monitor by pulse oximetry; intubate if there is obtundation, seizures, or a depressed gag reflex.
 - (b) Establish IV access of normal saline. Young, healthy patients with exertional heat stroke are dehydrated and require aggressive fluid management. Those with classic heat stroke need IV fluids, but should not be rehydrated aggressively (fluid requirements are usually not large); rate of 250–300 mL/hr is generally adequate. Central venous pressure monitoring may be needed to guide fluid therapy in older patients and in those with cardiovascular disease.

- (c) Put the patient on a cardiac monitor. Tachydysrhythmias are common and respond to cooling (do not cardiovert); be careful not to insert the tip of the central venous pressure catheter into an irritable heart.
- (d) Establish continuous temperature monitoring via an esophageal or rectal probe.
- (e) Place a Foley catheter to monitor urine output.
- (f) Obtain baseline laboratory studies
 - i. Arterial blood gases (corrected for temperature), lactate
 - ii. CBC, glucose, electrolytes (including Ca^{++} and Mg^{++})
 - iii. BUN/creatinine, prothrombin time/INR and partial thromboplastin time, toxicology screen
 - iv. Thyroid and liver function tests
 - v. Urinalysis and urine myoglobin
 - vi. ECG and chest radiograph (especially in older patients)
- b. Medications
 - (1) Control shivering with a benzodiazepine or chlorpromazine as needed; induction of paralysis may be required.
 - (2) Antipyretics (aspirin, ibuprofen, acetaminophen) are not useful.
 - (3) Dantrolene has not been demonstrated to be effective.

III. COLD DISORDERS

A. Frostbite (local cold injury can occur at temperatures above and below freezing)

- 1. Pathophysiology
 - a. Causes of tissue damage
 - (1) Capillary stasis and thrombosis
 - (a) Cooling to 59°F (15°C) → maximal vasoconstriction and a significant decrease in cutaneous blood flow
 - (b) Continued cooling to 50°F (10°C) → cold-induced vasodilation (the "hunting response") in which vasoconstriction is interrupted in a cyclical fashion by periods of vasodilation in an effort to protect the extremity from the cold
 - (c) When returning cold blood starts to decrease the core body temperature, blood flow to the extremity shuts down and the tissue temperature drops further → capillary endothelial damage → increased platelet affinity and plasma leakage from the intravascular space → increased viscosity, RBC stasis, and vessel thrombosis; frostbite occurs when the tissue temperature drops below 32°F (0°C).
 - (2) Ice crystal formation in tissues
 - (a) Extracellular ice contributes to intracellular dehydration and destruction of protein.
 - (b) Intracellular ice destroys cell architecture and function.
 - (3) Reperfusion injury: rewarming → return of blood flow to the injured extremity and release of arachidonic acid metabolites (thromboxane, prostaglandins) from the damaged endothelial cells → vasoconstriction, platelet clumping, and sludging of WBCs → progressive tissue loss

- b. Most likely sites of tissue damage because they are farthest from the body core: hands, feet, ears, face, and nose
 - c. Factors that affect the severity of tissue injury
 - (1) Temperature and duration of cold exposure: remember that wet skin and clothing freeze faster than dry skin and clothing.
 - (2) Humidity: adds to correct evaporative heat loss.
 - (3) Wind chill: moving air accelerates heat loss.
 - (4) High altitude: hypoxia affects the CNS and one's judgment ability; dehydration results from the increased respiratory rate → decreased blood flow
 - (5) Refreezing of a thawed extremity: greatly increases the severity of tissue damage and increases the likelihood of tissue loss
2. Clinical presentation
- a. Nonfreezing, wet → trench foot (immersion foot)
 - (1) Requires prolonged exposure (hours to days) to a wet environment
 - (2) Early symptoms include numbness, painful paresthesias, and leg cramps.
 - (3) Initial evaluation reveals a cold, pale, and anesthetic extremity.
 - (4) A hyperemic phase follows and is accompanied by severe burning pain.
 - (5) Tissue loss is uncommon.
 - b. Nonfreezing, dry → chilblains (pernio)
 - (1) Results from exposure to cold, damp air
 - (2) Lesions develop on exposed areas after a delay of 12–14 hours and are characterized by erythema, pruritus, and burning paresthesias.
 - (3) Women with Raynaud phenomenon are at highest risk.
 - c. Freezing, dry or wet → frostnip and frostbite
 - (1) Frostnip is an early response to cold exposure and is reversible.
 - (a) The first sign is pale, painful, itchy skin.
 - (b) If this progresses to frostbite, the skin remains cold to palpation and appears pale and gray.
 - (2) Frostbite
 - (a) Superficial: under the frozen surface, skin is soft.
 - i. Once thawed: large, clear, fluid-filled vesicles develop within 24–48 hours; when these are reabsorbed, the skin turns black and hard (carapace).
 - ii. The skin takes weeks to demarcate, and the carapace sloughs slowly, leaving new pink hypersensitive skin.
 - (b) Deep: under the frozen surface, skin is rock hard.
 - i. Once thawed, the area is cold and has a purple or red discoloration; small hemorrhagic vesicles and edema develop within 1–3 weeks.
 - ii. Weeks to months later, nonviable structures demarcate, become mummified, and slough off.
3. Management
- a. Prevention is the most important aspect of treatment. Proper clothing and nutrition, good health, and avoidance of fatigue and alcohol seem to be the most crucial aspects of prevention.

b. Frostnip

- (1) Rewarming may be initiated in the field (breath, body heat, other heat sources).
No rubbing.
- (2) Return to work is possible after rewarming, but the patient should be cautioned about recurring symptoms.

c. Frostbite

- (1) Rapid rewarming is key and should not be started until refreezing can be prevented.
- (2) Remove clothing and rewarm affected area in circulating water that is heated to 104°–107.6°F (40°–42°C); rewarming with dry heat can be dangerous because the frozen (insensitive) skin cannot detect heat, and burns may result.
- (3) Initiate general rewarming measures (as appropriate) and rehydration to treat the systemic hypothermia and dehydration that frequently occur in association with frostbite.
- (4) Provide tetanus prophylaxis and analgesia as needed.
- (5) Local care after full thawing
 - (a) Elevate the affected extremity.
 - (b) Debride or aspirate white and clear blisters; they contain chemical mediators of ischemia (eg, prostaglandin $F_{2\alpha}$ and thromboxane A₂) that can further damage underlying tissue.
 - (c) Leave hemorrhagic blisters intact, because debridement can result in extension of injury.
 - (d) Apply a topical thromboxane inhibitor (eg, aloe vera).
 - (e) Dress only open wounds and separate frostbitten digits with soft dressings; leave other frostbitten areas open to air.
 - (f) Avoid early surgical intervention.
- (6) Administer
 - (a) Ibuprofen 400 mg orally to inhibit the arachidonic acid cascade and promote fibrinolysis.
 - (b) Penicillin G 500,000 units IV to prevent streptococcal infection.

B. Hypothermia: core body temperature <95°F (35°C)**1. Pathophysiology****a. Normal body temperature regulation****(1) Heat loss**

- (a) **Conduction:** remember that the body loses heat 30 times faster in water than in air.
- (b) **Convection:** wind chill
- (c) **Radiation:** increased by peripheral vasodilation and decreased by insulated clothing
- (d) **Evaporation:** sweat, exhaled breath

(2) Heat gain and conservation

- (a) **Peripheral vasoconstriction**
- (b) **Increased metabolic rate**
- (c) **Shivering:** this ability is lost at 30°–32°C (86°–90°F).
- (d) **Behavior:** putting on clothes, going inside, etc

b. Body response to hypothermia

- (1) At 90°–95°F (32°–35°C), metabolic activity is in an excitation or responsive state and the body attempts to conserve as well as generate heat.
- (2) At temperatures <32°C (90°F), metabolic activity slows down or is in an adynamic stage and the body's compensatory mechanism fails; O₂ consumption and CO₂ production both decrease.

c. Effects of hypothermia on organ systems

(1) Cardiovascular

- (a) Blood pressure, pulse, and cardiac output all increase during the metabolic excitation phase (90°–95°F [32°–35°C]), but they all decrease once the temperature falls below 32°C (metabolic slowing phase).
- (b) ECG changes
 - i. Osborn or "J" waves
 - ii. T-wave inversion
 - iii. Prolonged PR, QRS, and QT intervals
 - iv. Dysrhythmias: risk develops when core temperature is <86°F (30°C); usual progression: sinus bradycardia → slow atrial fibrillation → ventricular fibrillation or asystole. The cold myocardium is very irritable, and ventricular fibrillation can be induced by rough handling or procedures. In addition, at core temperatures <77°F (25°C), spontaneous ventricular fibrillation and asystole may occur.
- (c) Decreased intravascular volume and increased blood viscosity can cause thrombosis and embolism.

(2) Pulmonary

- (a) The respiratory rate increases in the excitation phase and decreases in the slowing phase. As the tidal volume decreases and the gag/cough reflexes become depressed, there is significant risk of aspiration.
- (b) The oxyhemoglobin dissociation curve shifts to the left, thus decreasing oxygen delivery to tissues.
- (c) Arterial blood gases (ABGs)
 - i. Although the pH will be falsely low and the pO₂ and pCO₂ will be falsely high on an uncorrected ABG, the practice of correcting ABGs for temperature is both cumbersome and unnecessary.
 - ii. In addition, because the pH of neutrality is increased in hypothermia and hypothermic-induced abnormalities clear spontaneously with rewarming, it is unnecessary and potentially dangerous to correct any identified abnormalities.

(3) Gastrointestinal

- (a) Pancreatitis
- (b) Decreased hepatic function; this is important because drugs metabolized by the liver may reach toxic levels with usual doses, eg, lidocaine.

(4) Renal

- (a) Decreased concentrating ability → "cold diuresis" → volume depletion
- (b) Decreased renal blood flow and myoglobinuria → acute tubular necrosis

- (5) Endocrine function is generally well preserved; however, the serum glucose level may be high, low, or normal.

- (6) Acid-base disorders are common but variable; acidosis or alkalosis may occur.
- (7) CNS: As hypothermia progresses, the level of consciousness becomes more and more depressed; mild incoordination → confusion → lethargy → coma (pupils may become dilated and unreactive). Associated with these changes are a progressive decrease in cerebral blood flow and an even greater decrease in cerebral oxygen requirements, which may provide a "brain protective" effect against anoxia and ischemia.
- 2. High-risk patients
 - a. The elderly lose their ability to generate heat and sense cold.
 - b. The very young conserve heat poorly because of high surface area to mass ratio and inadequate subcutaneous tissue.
 - c. Those individuals with an altered sensorium due to CNS disease, alcohol, or drugs
- 3. Predisposing factors
 - a. Environmental exposure
 - (1) Nonimmersion hypothermia: most common presentation
 - (2) Immersion hypothermia: water temperature <60°–70°F (16°–21°C)
 - b. Metabolic
 - (1) Hypoglycemia from any cause
 - (2) Decreased metabolic rate
 - (a) Hypopituitarism
 - (b) Hypothyroidism
 - (c) Hypoadrenalism
 - c. CNS or hypothalamic dysfunction
 - (1) Head trauma
 - (2) Brain tumor
 - (3) Stroke
 - (4) Wernicke encephalopathy
 - d. Drug-related
 - (1) Alcohol is number one!
 - (2) Barbiturates
 - (3) Benzodiazepines
 - (4) Narcotics
 - (5) Phenothiazines
 - (6) Cyclic antidepressants
 - (7) Excess insulin (when it causes hypoglycemia)
 - e. Any acute debilitating condition (diabetic ketoacidosis, traumatic injuries, etc)
 - f. Skin disorders, burns, and exfoliative dermatoses cause increased evaporation and decreased ability to vasoconstrict.
 - g. Sepsis/myxedema coma
- 4. The diagnosis may not be obvious, especially in temperate settings. Be alert for risk factors and use a rectal temperature probe or other special low-reading thermometer to establish the diagnosis; standard clinical thermometers only read down to 95°F (35°C).

5. Management

a. General measures

- (1) Handle the patient gently to avoid precipitating ventricular fibrillation; keep the patient in a horizontal position to avoid inducing or worsening hypotension via orthostatic mechanisms.
- (2) ABCs and stabilization
 - (a) Indications for intubation are the same as those for normothermic patients.
 - (b) Warmed humidified oxygen (107.6°–114.8°F [42°–46°C]) and warmed IV fluids (109.4°F [43°C]) should be administered if the patient is moderately (86°–93.2°F [30°–34°C]) to severely hypothermic (<86°F [30°C]).
 - (c) Check blood glucose level.
 - (d) Monitoring
 - i. Cardiac (including an ECG)
 - ii. Ins and outs (Foley catheter if not alert)
 - iii. Core temperature
- (3) Treat only life-threatening dysrhythmias (ventricular fibrillation, asystole), because the rest correct with rewarming; besides, all therapeutic modalities have unpredictable results in this setting.
 - (a) Avoid atropine and lidocaine (ineffective in hypothermia).
 - (b) In patients with ventricular fibrillation, administration of MgSO_4 produces spontaneous defibrillation.
- (4) Antibiotics, steroids, and thyroxine should be given only for specific indications. Remember that most myxedema comas are triggered by underlying infections.

b. Rewarming: the best method is still controversial.

(1) Passive external rewarming

- (a) Place the patient in a warm environment, remove wet clothes, and prevent further heat loss by covering with a blanket.
- (b) This is safe but slow and is most appropriate if the hypothermia developed slowly and has been present for some time.

(2) Active external rewarming

- (a) Apply heat to the body surface with one of the following:
 - i. Forced-air rewarming (eg, temperature management blanket)
 - ii. Warming blanket
 - iii. Immersion in warm water
 - iv. Radiant heat
- (b) This is effective but has potential problems.
 - i. Cold blood returning from the periphery causes further cooling ("core temperature afterdrop"), brings lactic acid with it ("rewarming acidosis"), and causes a relative hypovolemia from peripheral vasodilation ("rewarming shock").
 - ii. Metabolic demands in the periphery exceed perfusion capabilities. These complications can be minimized by applying the heat source to the trunk only, and by using this modality in combination with one or more of the active core rewarming methods described below.
 - iii. Patients who are submerged in warm water are difficult to monitor.

- (3) Active core rewarming
 - (a) Noninvasive methods
 - i. Inhalation rewarming: warm humidified oxygen by mask or endotracheal tube at 107.6°–113°F (42°–45°C)
 - ii. Heated IV fluids (D5NS is the preferred solution)
 - (b) Invasive methods
 - i. Gastric or colonic lavage with warm saline: rarely needed or used
 - ii. Bladder lavage with warm saline using a Foley catheter: very poor heat transfer
 - iii. Peritoneal lavage
 - iv. Extracorporeal rewarming: usually accomplished by hemodialysis, cardiopulmonary bypass, or arteriovenous or venovenous rewarming
 - v. Closed thoracic lavage: warm saline is infused through two thoracostomy tubes.
 - (c) The advantage of active core rewarming is that preferential rewarming of the heart and internal organs results in improved function and avoidance of problems associated with peripheral vasodilatation.
 - (d) Active core rewarming is the modality of choice for rewarming patients with severe hypothermia (core temperature <86°F [30°C]) and those with cardiac instability.
- (4) Choice of rewarming method depends on the severity of the hypothermia, its cause and duration, and the patient's status on presentation to the emergency department. Guidelines follow:
 - (a) Use multiple methods.
 - (b) Patients who are still in the excitation phase probably do not need active rewarming.
 - (c) Patients with life-threatening cardiac dysrhythmias need rapid rewarming; use both noninvasive and invasive active core rewarming techniques.
- (5) Outcome: the underlying or precipitating disease process and its severity are more important predictors of outcome than the degree of hypothermia on presentation.
- (6) Hypothermic patients should not be pronounced dead until after they have been rewarmed to 95°F (35°C) and subsequent resuscitative efforts are still unsuccessful. "No one's dead until they're warm and dead." However, consider terminating resuscitation earlier for refractory cardiovascular unresponsiveness in patients with asphyxia (submersion or avalanche), lethal injuries, or serum K⁺ >10 mEq/L. (See also hypothermia in the Dysrhythmia section, where the treatment focus is entirely on the degree of hypothermia and the patient's cardiovascular status, page 11.)

IV. BITES AND STINGS

A. Human bites

1. The most common aerobic pathogens are *Streptococcus* and *Staphylococcus aureus*. Most infections are polymicrobial and often involve both aerobes and anaerobes, especially *Eikenella corrodens*.

2. The intact skin surrounding these bites should be cleansed with a broad-spectrum antimicrobial agent such as povidine-iodine; if there is a history of sensitivity/allergy to iodine, chlorhexidine may be used (but it is less effective and known to cause corneal ulcers if comes into contact with the eyes). The wound itself should be meticulously cleansed with copious amounts of sterile saline solution using a high-pressure irrigation technique.
3. Inspect the wound for tooth fragments (from "fight bites") and sharply debride devitalized tissues. Hand wounds should be radiographed to exclude fractures, foreign bodies, and air in the joint spaces.
4. Suturing
 - a. Wounds of the head, face, and neck may be sutured (primary closure) if the patient presents within 24 hours of injury and if there is no evidence of infection; if signs of infection are evident or the wound is >24 hours old, it should be left open.
 - b. All hand wounds should be left open initially; this includes closed fist/"fight bite" injuries (in which the extensor tendon and its sheath can be inoculated).
 - c. Most wounds to other areas may be sutured as long as there is no evidence of infection or delay in treatment.
5. Antibiotics are indicated for all human bites
 - a. High-risk conditions
 - (1) All hand wounds (strong evidence supporting infection prevention)
 - (2) Any wound associated with comorbidity
 - (a) Diabetes
 - (b) Asplenia
 - (c) Immune deficiency
 - (d) Bite inflicted by a hospitalized or institutionalized person
 - (3) Any wound with a poor blood supply, eg, anterior shin
 - b. Wound type (laceration, puncture) does not influence the effectiveness of antibiotic prophylaxis (ie, same for cat, dog, and rabbit bites)
 - c. Parenteral (IM or IV) antibiotics and possible admission
 - (1) Wounds >24 hours old that are obviously infected
 - (2) Wounds involving tendon, joint, or bone
 - (3) Signs of systemic infection (tachycardia, fever, extensive cellulitis, lymphangitis, etc)
 - d. Regimens for prophylaxis/treatment (3–5 days)
 - (1) Provide coverage for *S aureus*, *Streptococcus* spp, *Pasturella*, *Bacteroides*, and *Eikenella corrodens*
 - (2) *E corrodens* (a likely pathogen in hand wounds) is resistant to many agents including dicloxacillin, many first-generation cephalosporins, clindamycin, and the aminoglycosides.
 - (3) Acceptable regimens (first two are best choices)
 - (a) A second or third-generation cephalosporin (eg, cefuroxime, cefoxitin)
 - (b) Dicloxacillin plus ampicillin or penicillin
 - (c) Amoxicillin-clavulanate
 - (d) Clarithromycin or clindamycin and TMP-SMX or doxycycline (in penicillin-allergic patients)

- (4) An aminoglycoside (eg, gentamicin) should be added to one of the above regimens in patients with diabetes, because these patients have a higher incidence of infection with gram-negative bacilli.
6. Other appropriate measures
 - a. Provide tetanus prophylaxis as indicated.
 - b. Consider administration of hepatitis B vaccine and immunoglobulin and testing for HIV when appropriate.

B. Dog bites

1. Remember the mnemonic "RATS"

Rabies

Antibiotics

Tetanus

Soap

2. Common pathogens include α -hemolytic streptococci, *S aureus*, and (less often) *Pasteurella multocida*. A potentially lethal infection is caused by the organism *Capnocytophaga canimorsus* and occurs most often in immunocompromised patients (particularly the splenectomized and those with cirrhosis); mortality is secondary to sepsis, disseminated intravascular coagulation, or meningitis.
3. Most lacerations (especially facial) that are seen within a few hours of injury should be closed primarily after meticulous irrigation and debridement.
4. Wounds >12 hours old and hand wounds should be left open initially with follow-up in 3–5 days for delayed primary closure; the exception is facial wounds, which should be closed primarily.
5. Management
 - a. Antibiotic regimens
 - (1) Prophylaxis with cephalexin, dicloxacillin, or amoxicillin-clavulanate (erythromycin or TMP-SMX in the penicillinallergic patient) is indicated for all hand wounds and recommended for all puncture wounds, lacerations, and patients with comorbid conditions.
 - (2) Treatment for infected wounds is determined by the probable pathogen involved, as follows:
 - (a) Cephalexin, dicloxacillin, or amoxicillin-clavulanate (erythromycin or TMP-SMX in the penicillin-allergic patient) is indicated for infections occurring >24 hours after injury (*Streptococcus* or *Staphylococcus* are most likely pathogens).
 - (b) Penicillin VK (TMP-SMX, ciprofloxacin, or clarithromycin in the penicillin-allergic patient) is indicated for wound infections occurring within 24 hours of injury (*Pasteurella multocida* most likely pathogen).
 - (c) Penicillin or a cephalosporin (erythromycin in the penicillinallergic patient) is appropriate for suspected infection with *C canimorsus*.
 - i. Immunocompromised patient who presents with gangrene or severe systemic manifestations such as sepsis, endocarditis, or cardiopulmonary or acute renal failure
 - ii. Because this is a potentially life-threatening infection, highrisk patients should be given prophylactic treatment.
 - b. Indications for admission and empirical, parenteral antibiotic therapy with penicillin G and nafcillin (pending culture results)

- (1) Wound infection plus any of the following:
 - (a) Lymphangitis/lymphadenitis
 - (b) Tenosynovitis
 - (c) Septic arthritis
 - (d) Osteomyelitis
- (2) Systemic signs (fever, tachycardia, etc)
- (3) Injury to tendons, joints, or bone
- c. Tetanus prophylaxis should be administered as indicated.
- d. Rabies prophylaxis should be considered but is rarely indicated in the United States.

C. Cat bites

- 1. Most common pathogen is *Pasteurella multocida*; other, less commonly encountered organisms, are similar to those associated with dog bites (including *C caninorsus*).
- 2. Wound closure
 - a. Puncture wounds and lacerations <1–2 cm long should be left open, because they cannot be cleaned well; those in cosmetically important areas should be followed-up with delayed primary closure.
 - b. The wound should be checked within 24 hours.
- 3. Antibiotics
 - a. Prophylaxis is indicated for patients with:
 - (1) Hand wounds
 - (2) Arthritis
 - (3) Prosthetic joints/valves
 - (4) Immunocompromise
 - (5) Puncture wounds and lacerations
 - b. The antibiotic regimen chosen should cover *P multocida* (which is exquisitely sensitive to penicillin but resistant to erythromycin and cephalexin) and *S aureus*.
 - c. Acceptable regimens
 - (1) Penicillin plus dicloxacillin
 - (2) Ampicillin-clavulanate
 - (3) TMP-SMX (in penicillin-allergic patients)
 - (4) Ciprofloxacin or cefuroxime (in penicillin-allergic patients)
 - d. Indications for admission and empirical parenteral antibiotic therapy are the same for dog bites; use penicillin G and nafcillin.
 - e. Tetanus toxoid and rabies prophylaxis should be provided as indicated.
- 4. Cat scratch disease
 - a. Etiology and epidemiology
 - (1) Most commonly affects children and adolescents
 - (2) Causative organism is *Bartonella henselae*, a small, pleomorphic, gram-negative bacillus
 - (3) Incubation period is ~1 week (range 3 days to 6 weeks).
 - b. Clinical presentation: characterized by persistent regional lymphadenitis that is often unilateral and typically involves the lymph nodes of the arms or legs

c. Management

- (1) Most cases resolve spontaneously over a period of weeks to months and require only symptomatic treatment.
- (2) Antibiotics are usually reserved for patients with severe or persistent disease. Acceptable choices include TMP-SMX, gentamicin, ciprofloxacin, and erythromycin.

D. Venomous snake bites

1. Most venomous snake bites in the United States (>98%) are inflicted by pit vipers, and 95% of the bites involve the extremities.
2. Management
 - a. The mainstay of therapy is administration of antivenin IV (described below).
 - b. Establish monitoring and begin aggressive supportive therapy as appropriate.
 - c. Immobilize the affected extremity to delay systemic absorption of venom; application of a loosely constricting band proximal to the bite site within 30 minutes of envenomation may also be helpful. Local therapy (mechanical, suction, wound excision/incision, elastic wrap) is not currently recommended.
 - d. Cleanse the wound, and administer tetanus prophylaxis (if indicated).
3. Pit vipers (Crotalidae family)
 - a. Crotalid venom produces both local and systemic effects; its major effects occur in local tissue, blood vessels, and blood components.
 - b. The severity of envenomation and the severity of the bite (rattlesnake > cottonmouth > copperhead) determine both the need for and the amount of antivenin to be administered.

Table 34: Clinical Parameters Used to Assess Need for and Amount of Antivenin

Parameter	Dry Bite	Minimal	Moderate	Severe
Signs	Puncture wound ± swelling	Swelling, erythema, ecchymosis	Evidence of spreading	Entire area or limb involved
Symptoms				
Focal	Slight pain	Minimal pain	Progressive pain	Severe pain
Systemic	None	None or minimal	Present	Fulminant
Coagulation tests	Normal	Normal	Abnormal but no bleeding	Abnormal and bleeding

- c. The antivenin Crotalidae Polyvalent Immune Fab Ovine not only demonstrates clinical efficacy but appears to be associated with fewer allergic symptoms and reduced incidence of serum sickness. However, because it has a short half-life, venom effects can recur, and a repeat dose regimen must be used.
 - (1) Minimal to moderate signs/symptoms: 4–6 vials diluted in 100 mL normal saline administered IV over 1 hour
 - (2) Moderate to severe signs/symptoms: 6 vials diluted in 100 mL normal saline administered IV over 1 hour
 - (3) Progression of signs/symptoms: 2 vials in 100 mL normal saline administered IV over 20–30 minutes
 - (4) Recurrent signs/symptoms 2 vials in 100 mL normal saline every 6 hours × 3 (6 vials total)

- d. Patients with dry bites (no signs/symptoms of envenomation and normal laboratory studies) may be discharged after 8–12 hours of observation (possibly longer depending on the species); all others should be admitted. One of every five bites is a dry bite.
- 4. Coral snakes (Elapidae family)
 - a. Coral snakes may be confused with king snakes and milk snakes. Coral snakes have wider bands of black and have red directly next to a yellow stripe. Remember “Red on yellow, kill a fellow. Red on black, venom lack.”
 - b. Elapid venom is largely neurotoxic; it has a curare-like effect on neuromuscular transmission and can produce total flaccid paralysis. It does not cause local edema.
 - c. Clinical presentation
 - (1) Signs and symptoms of envenomation may be delayed up to 12 hours.
 - (2) A change in mental status and cranial nerve dysfunction (eg, diplopia) are the earliest findings to develop.
 - (3) Respiratory paralysis is the major life threat.
 - d. Antivenin is available and should be administered early (even in the absence of any signs or symptoms) in patients who clearly have been bitten.
 - (1) Because the antivenin is equine-derived, precautions for a possible allergic reaction during administration must be observed. However, in the case of a positive skin test, antivenin should be withheld because these patients do well with aggressive supportive measures (eg, prolonged intubation and ventilation) alone.
 - (2) Begin with 3 vials; administer 3–5 additional vials if signs and symptoms develop or worsen.
 - e. All patients with suspected or known envenomation should be admitted for observation or definitive care.
 - f. Delayed onset of serum sickness, 1–2 weeks after treatment, is the most frequent reaction to both types of antivenin.
- E. Bee stings, ant bites, and other insect bites and stings
 - 1. Immediate concerns: upper airway obstruction, anaphylaxis, and toxic reactions from multiple stings
 - 2. If stingers are present, remove them immediately by whatever means possible. While it has been traditionally recommended that stingers be removed by scraping rather than forceps, evidence refutes this concern.
- F. Black widow spider (*Latrodectus mactans*)
 - 1. The female spider is more poisonous than the male. The female has a red hourglass shape on the thorax. The male is much smaller and has a blue, yellow, or white hourglass on the thorax.
 - 2. Clinical presentation (symptoms often wax and wane)
 - a. Local → a dull, muscle cramping sensation
 - (1) Upper extremity bite: chest pain
 - (2) Lower extremity or genitalia bite
 - (a) Abdominal pain that may simulate an “acute abdomen”; a differentiating feature is the presence of abdominal rigidity with little or no associated tenderness.
 - (b) Rectal spasm with normal bowel sounds

- b. Systemic → neurotoxic effects (diffuse CNS and peripheral nervous excitation)
 - (1) Diffuse muscle pain and stiffness
 - (2) Dizziness and restlessness
 - (3) Profuse sweating (which may be uniquely focal to one area) and weakness
 - (4) Difficulty in speaking and ptosis
 - (5) Hypertension and tachycardia
 - 3. Management
 - a. First aid: ice pack to the bite area
 - b. Treatment
 - (1) Cleanse with soap and water (look for fang marks and halo lesions)
 - (2) Tetanus toxoid
 - (3) Disposition
 - (a) 1 hour if the spider not positively identified and the patient remains symptom-free during this time
 - (b) 4 hours if the spider positively identified but the patient remains symptom-free during this time
 - c. Specific therapeutic measures
 - (1) Signs and symptoms may be treated with:
 - (a) Narcotic analgesics (the mainstay of therapy and pain relief)
 - (b) Benzodiazepines (relieve muscle spasms)
 - (2) Use of antivenin (one vial is usually sufficient)
 - (a) Antivenin is not used in all cases because:
 - i. The effects of black widow spider bites are self-limited with a low mortality rate; because use of antivenin has been associated with death, most patients are not given antivenin.
 - ii. The antivenin is equine-derived (pretesting for horse serum sensitivity is advised) and can therefore produce anaphylaxis. Serum sickness can also occur but is uncommon, because so little antivenin is used.
 - iii. The antivenin, when combined with β -adrenergic blockers, can produce anaphylactic reactions that are refractory to treatment.
 - (b) Indications for antivenin
 - i. Patients <16 years or >65 years old
 - ii. Patients with severe pain (despite analgesics) or severe envenomations
 - iii. Patients unable to stand the stress of the envenomation (due to concurrent illness)
 - iv. Patients with dangerous hypertension
 - v. Pregnant women
- G. Brown recluse spider (*Loxosceles reclusa*)**
- 1. The brown recluse is the size of a penny, and the head has a fiddle-shaped black marking.
 - 2. Clinical presentation
 - a. Local pain develops at the site in 3–4 hours.
 - (1) The lesion starts with a halo of vasoconstriction, which then develops a central bleb (the classic bull's-eye lesion), which may continue to spread over several days.
 - (2) Tissue necrosis is a major complication that may require surgical consultation.

- b. Systemic (loxoscilism): thought to be an allergic reaction
 - (1) Develops over 24–72 hours
 - (2) Fever, chills, petechial rash, nausea, vomiting, weakness
 - (3) May progress to hemolysis, shock, renal failure, disseminated intravascular coagulation
 - (4) Most fatalities are in children.
3. Local tissue necrosis is a major complication that may require surgical consultation.
4. Management
 - a. Wash wound with soap and water.
 - b. Apply ice compresses locally to decrease pain and diminish the evolution of cutaneous inflammation.
 - c. Administer tetanus toxoid.
 - d. Observe the patient in the emergency department if elapsed time since the bite is <6 hours.
 - e. Specific measures
 - (1) Use of diamino-diphenyl sulfone (a leukocyte-esterase inhibitor) 100 mg orally bid is controversial but may be useful in treating the local effects of the venom; it is also contraindicated in patients with glucose-6-phosphate dehydrogenase deficiency.
 - (2) Erythromycin 250 mg orally qid if infection exists
 - (3) Antihistamines are useful in treating associated pruritus.
 - (4) Patients with significant systemic reactions should be hospitalized. Administration of parenteral corticosteroids prevents hemolysis; however, they have no effect on the tissue necrosis.
 - (5) Antivenin is not currently available.

H. Scorpion stings

1. Envenomations are graded from I to IV (mild to severe) based on the clinical findings present, as follows:
 - a. Grade I: localized pain/paresthesias
 - b. Grade II: pain/paresthesia remote from site
 - c. Grade III: cranial nerve or somatic neuromuscular dysfunction
 - d. Grade IV: cranial nerve and somatic skeletal neuromuscular dysfunction
2. Management
 - a. All patients should receive local wound care and tetanus toxoid (if indicated) and be observed for evidence of progression.
 - b. Further treatment is determined by the grade of envenomation.
 - (1) Symptomatic therapy (ice packs to area, analgesics, sedation) alone is adequate for grades I and II.
 - (2) Antivenin may be required for grades III and IV; the antivenin is goat-serum derived, carries the risk of anaphylaxis and serum sickness, and is available only in Arizona (the Arizona bark scorpion is the most deadly scorpion in North America); skin testing is advised.

I. Jellyfish stings

1. Jellyfish envenomate their victims via stinging cells called nematocysts (cnidocysts).
2. Victims of many severe toxic jellyfish stings (especially the Indo-Pacific box jellyfish) do not survive to shore; some succumb to the direct effects of the venom, but many drown due to the cardiopulmonary complications from the envenomation. Problems with survivors include removal of nematocysts, pain control, and prevention of infection.
3. Treatment is controversial and unproven (therefore, less likely to be on the exam).
 - a. Remove patient from the water.
 - b. Wash the area with sea water; do not use fresh water, because it may cause the nematocysts to discharge, which may then cause severe pain.
 - c. Do not rub the area with sand, because this worsens the condition.
 - d. Remove tentacles with forceps or a thickly gloved hand.
 - e. Any nematocysts that remain after washing should be fixed by pouring vinegar (or isopropyl alcohol) over the wound area for at least 30 minutes. Next, dust the area with talcum powder or cover with shaving cream. The nematocysts, now adherent to the powder or cream, can be scraped off with a knife or razor blade.
 - f. Wash the area with sea water once again and then apply a topical anesthetic/antihistamine or low-potency steroid cream (which may be soothing).
 - g. Administer tetanus toxoid.
 - h. Allergic reactions are common and should be treated in the usual manner.
4. Hospitalize patients who sustain severe envenomation or develop an allergic reaction.

V. ALTITUDE DISORDERS

A. High-altitude disorders (can be avoided by proper acclimatization or use of acetazolamide 1–2 days before ascent)

1. Acute mountain sickness

a. Clinical presentation

- (1) Onset of symptoms is within a few hours; duration is 3–4 days.
- (2) Symptoms include bifrontal headache, anorexia, nausea, and sleeping difficulties. The headache is usually worsened with the Valsalva maneuver and with stooping over; it is most severe during the night and in the morning on awakening.

b. Management

- (1) Symptomatic treatment and a halting of the ascent for 12–36 hours until acclimatization has occurred is usually all that is needed in mild cases.
- (2) Effective agents include NSAIDs or acetaminophen for headache and prochlorperazine for nausea and vomiting. Prochlorperazine also has the advantage of increasing the hypoxic ventilatory response.
- (3) Supplemental low-flow oxygen is useful in relieving symptoms, especially at night, during sleep.
- (4) Acetazolamide 125–250 mg orally bid speeds acclimatization and is also effective for treatment of acute mountain sickness; it is contraindicated in patients allergic to sulfa.

- (5) **Dexamethasone 4 mg orally, IM, or IV qid is also effective for prophylaxis as well as treatment of acute mountain sickness but, because of adverse effects and problems with rebound when the drug is stopped, it is recommended only as treatment (not prophylaxis) and is reserved for moderate to severe cases.**
- (6) **Descent is the definitive therapy and is rapidly effective but is not necessary unless symptoms progress or are prolonged and debilitating.**
- 2. **High-altitude pulmonary edema (HAPE)**
 - a. This is noncardiogenic (wet lungs and normal heart size) in origin; it should not be confused with CHF.
 - b. Clinical presentation
 - (1) Onset of signs and symptoms is 1–4 days after ascent and often preceded by exertion.
 - (2) Symptoms include weakness, headache, cough, shortness of breath, cyanosis, and rales.
 - c. Management
 - (1) Bedrest, oxygen, increased positive-airway pressure (via CPAP, BiPAP, or endotracheal intubation) and descent
 - (2) If descent is impossible:
 - (a) The Gamow bag (portable hyperbaric chamber) simulates descent to a lower altitude.
 - (b) Nifedipine may be helpful, because it reduces pulmonary artery pressure through its vasodilatory effect.
- 3. **High-altitude cerebral edema (HACE)**
 - a. Can result in permanent neurologic injury or even death
 - b. Clinical presentation
 - (1) Common signs/symptoms: confusion, ataxia, retinal hemorrhages, hallucinations, and headache
 - (2) Can progress to coma if untreated
 - c. Management
 - (1) Oxygen, head elevation, descent, and dexamethasone; hyperbaric therapy is also effective if available.
 - (2) Loop diuretics (eg, furosemide, bumetanide) may be helpful in the treatment of both HAPE and HACE, but hypoperfusion is a significant risk so they should be used cautiously, if at all.

B. Dysbarism

1. Barotrauma of descent ("squeeze")

- a. **Symptoms develop because of compression of air that is trapped in various parts of the body during descent. Divers can develop symptoms in water as shallow as 4.5–6.5 feet.**
- b. **Barotitis externa ("external ear squeeze")**
 - (1) **Occurs when the external auditory canal is occluded (eg, by cerumen, foreign bodies, exostoses, ear plugs, or a too-tight fitting wet suit hood)**
 - (2) **Compression of the enclosed air with descent produces pain and/or bloody otorrhea (from blood-filled cutaneous blebs along the canal or tympanic membrane rupture).**

- (3) Management: keep canal dry; no diving (or swimming) until healed.
 - c. Barotitis media ("middle ear squeeze")
 - (1) Most common type of aural barotrauma
 - (2) Results from failure to equalize the pressure between the middle ear and the water; this occurs because of occlusion or dysfunction of the eustachian tube.
 - (3) Predisposing factors
 - (a) Mucosal congestion secondary to an upper respiratory infection, allergies, or smoking
 - (b) Mucosal polyps
 - (c) Excessively vigorous autoinflation maneuvers
 - (d) Previous maxillofacial trauma
 - (4) As the pressure differential increases, the diver experiences ear fullness or pain (which will worsen until the tympanic membrane ruptures, unless the dive is aborted); if the tympanic membrane does rupture, the diver may become disoriented because of severe nausea and vertigo that result from the caloric stimulation of cold water entering the middle ear.
 - (5) The severity of the injury can be assessed from the amount of hemorrhage associated with the eardrum; a grading system may be used, with "0" signifying no hemorrhage (only symptoms) to "5" indicating tympanic membrane rupture with gross hemorrhage.
 - (6) Management: no diving and nasal decongestants until healed; if the tympanic membrane is ruptured, antibiotics are indicated; resolution occurs within 1 week.
 - d. Barotitis interna ("internal ear squeeze")
 - (1) Permanent injury to the cochleovestibular system may occur.
 - (2) Results from sudden pressure differences between the internal and middle ear, which may occur after an overzealous Valsalva maneuver or extremely rapid descent.
 - (3) Clinical presentation: classic symptomatology → tinnitus, deafness, and vertigo
 - (4) Mechanisms of injury: inner ear hemorrhage, rupture in Reissner membrane → mixing of endolymph and perilymph, round or oval window fistulation, or a combination of these insults
 - (5) Management: bed rest with the head elevated, sedentary activities as tolerated, symptomatic therapy, and ENT referral; early surgical intervention is indicated for patients with total (or near total) hearing loss.
 - e. "Sinus squeeze"
 - (1) Sensation of fullness, pressure, or pain in the affected sinuses or hemorrhage
 - (2) The maxillary and frontal sinuses are most often affected.
 - (3) Antibiotics are usually indicated if the frontal sinuses are involved; otherwise, the treatment is the same as for barotitis media.
2. Barotrauma of ascent
- a. Symptoms develop because of expansion of air that is trapped in bodily spaces during ascent.
 - b. Examples
 - (1) Middle ear and sinus barotrauma of ascent ("reverse squeeze")
 - (2) Barodontalgia

- (3) Aerogastralgia (air in the gut)
- (4) Pulmonary barotrauma
 - (a) Mediastinal or subcutaneous emphysema (most common)
 - (b) Pulmonary interstitial emphysema
 - (c) Alveolar hemorrhage
 - (d) Pneumothorax
 - (e) Pulmonary air (arterial gas) embolism
 - i. The most severe complication of pulmonary barotrauma
 - ii. Immediately after ascent, air bubbles enter the systemic circulation through ruptured pulmonary veins.
 - iii. Arterial occlusion from an air embolus can occur at any site, including the coronary arteries (MI, cardiac arrest). However, the most common presentation is a neurologic event, eg, level of consciousness, blindness, deafness, vertigo, confusion, seizures, monoplegia, or asymptomatic multiplegia.
 - iv. Symptoms develop within 10 minutes of surfacing from a dive, and often much sooner.
 - v. Treatment of air embolism (pulmonary, coronary, or cerebral) is recompression in a hyperbaric chamber as soon as possible. During transport, the patient should be given oxygen and placed in the supine position. Placement in Trendelenburg is no longer recommended, because it may worsen cerebral edema and respiratory distress.
- 3. Barotrauma of descent or ascent is the result of a direct effect of pressure changes on the body. Nitrogen narcosis and decompression sickness, on the other hand, are the result of an indirect effect of pressure changes on the body.
 - a. Barotrauma occurs secondary to the compression or expansion of air in bodily spaces.
 - b. Nitrogen narcosis and decompression sickness occur secondary to breathing gases (usually nitrogen) at higher than normal atmospheric pressure.
 - (1) Nitrogen narcosis occurs as the diver descends to depths >70 feet. The clinical picture resembles alcoholic intoxication and can impair judgment; the condition clears with resurfacing.
 - (2) Decompression sickness occurs if the diver ascends too quickly.
 - (a) Symptoms result from dissolved inert gas (nitrogen) bubbles reentering tissues and blood vessels; it can involve any organ system.
 - i. Cutaneous → "skin bends"
 - ii. Musculoskeletal, most frequently affected → "the bends"
 - iii. Pulmonary → "the chokes"
 - iv. CNS
 - Spinal cord, most frequently affected → acute paraplegia
 - Cerebellar → "the staggers"
 - Cerebral
 - (b) Treatment is recompression in a hyperbaric chamber as soon as possible. During the transport, 100% oxygen should be administered to provide a favorable gradient for nitrogen washout and improve oxygenation of injured tissues.

VI. SUBMERSION INJURY

A. Definitions

1. At the 2002 World Congress on Drowning, a consensus was reached to redefine drowning to improve the accuracy of analysis and comparison of studies done on the subject.
2. Drowning is defined as a process resulting in primary respiratory impairment from submersion in a liquid medium. A liquid-air interface must be present in the victim's airway.
3. The outcome of drowning may include:
 - a. Delayed morbidity
 - b. Delayed death
 - c. Life without morbidity
4. The following terms were discarded:
 - a. Wet drowning
 - b. Dry drowning
 - c. Near drowning
 - d. Passive drowning
 - e. Active drowning
 - f. Secondary drowning
 - g. Silent drowning
5. Immersion syndrome is sudden death after submersion in very cold water (presumed to be due to vagally mediated asystole or ventricular fibrillation).
6. Postimmersion syndrome is delayed deterioration of a patient who is initially relatively asymptomatic; deterioration is generally due to respiratory insufficiency and may be delayed several hours to several days (ARDS). This is the major complication of submersion injury.

B. Epidemiology

1. Drowning is the third most common cause of accidental death in the United States, claiming 8,000–9,000 lives/year, with brain injury being the major cause of death from drowning.
2. Freshwater submersion injury (particularly in swimming pools) is more common than saltwater submersion injury, but the type of water has no bearing on morbidity and mortality.
3. Age distribution is bimodal, with peaks occurring in children <5 years old (consider child abuse) and teenagers.
4. Males predominate in every age group.
5. Risk factors
 - a. Males
 - b. Age <5 years old
 - c. Drugs (particularly alcohol)
 - d. Inadequate swimming skills
 - e. Exhaustion
 - f. Hyperventilation before underwater swimming

g. Trauma

- (1) Accidental (cervical spine injuries, head injuries)
- (2) Nonaccidental (child abuse)

h. Underlying illnesses

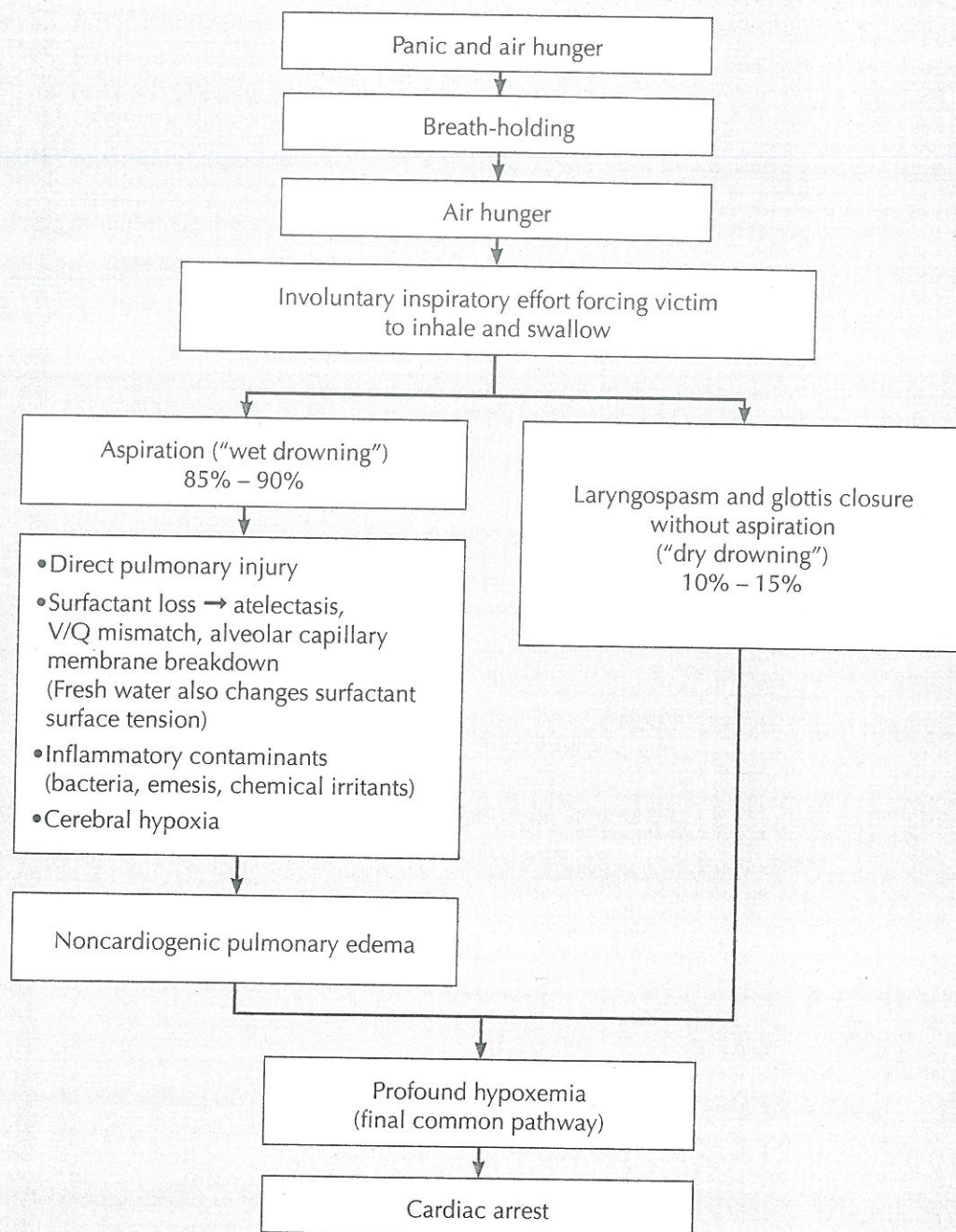
- (1) Hypoglycemia
- (2) Seizures
- (3) Myocardial infarction
- (4) Cardiac dysrhythmias
- (5) Depression/suicide attempt

i. Lack of supervision

j. Hypothermia

C. Pathophysiology

1. Sequence of events (are essentially the same for freshwater and saltwater submersion)



Sequence of Events in Submersion Injury

2. Other pathophysiologic characteristics of submersion injury
 - a. Metabolic acidosis (lactic acidosis type A) develops in many patients and is due to hypoxemia (anaerobic metabolism) and poor perfusion.
 - (1) It is usually well tolerated in children and may be reversed with adequate ventilation and oxygenation.

- (2) It is less well tolerated in adults.
 - (a) In addition to ventilation and oxygenation, small doses of NaHCO_3 may be considered for refractory acidosis.
 - (b) Lactic acidosis persists if severe hypoxemia is not corrected.
- b. Most victims do not aspirate enough fluid to cause life-threatening changes in blood volume or serum electrolyte concentrations.
- c. Renal failure occurs infrequently in submersion injury.
 - (1) Acute tubular necrosis may develop secondary to hypoxemia.
 - (2) In freshwater submersion injury, acute renal failure may result from hemolysis and hemoglobinuria.
- d. Cerebral edema may develop (usually within 6–12 hours) from generalized neuronal death. Cerebral edema \rightarrow \uparrow intracranial pressure \rightarrow \downarrow cerebral perfusion pressure and \downarrow cerebral blood flow \rightarrow further ischemic neurologic injury

D. Prehospital care

- 1. Ineffective and potentially dangerous procedures
 - a. In-water CPR (but can do airway management in water)
 - b. Attempts to remove fluid from the lungs via postural drainage or the Heimlich maneuver
- 2. Initial assessment and stabilization
 - a. If patient is conscious, safer to throw flotation device to the victim than to jump in: "throw, tow, row, go."
 - b. Always start CPR in the pulseless, apneic victim even if there is only a remote possibility of success.
 - c. Airway maintenance and supplemental oxygen are of primary importance. An IV line should also be started if possible.
 - d. Immobilize the cervical spine and look for signs of spinal injury:
 - (1) Paradoxical respirations
 - (2) Unexplained hypotension
 - (3) Bradycardia
 - (4) Flaccidity
 - (5) Priapism
 - e. Prevent onset or worsening of hypothermia: remove wet clothes, dry the patient, and wrap in dry blankets.
- 3. Transport all submersion-injury victims to the hospital.

E. Emergency department

- 1. Remember that submersion injury is an airway problem first. Evaluate airway patency, adequacy of cervical spine immobilization, and mental status; naloxone and glucose are indicated if there is an altered level of consciousness.
- 2. Assess respiratory status, looking for signs of pulmonary insufficiency:
 - a. Tachypnea
 - b. Dyspnea
 - c. Use of accessory muscles
 - d. Wheezing, rales, or rhonchi
- 3. Provide supplemental oxygen.

- a. Always use 100% oxygen.
 - b. Avoid prolonged suctioning, steroids, prophylactic antibiotics, and early extubation.
 - c. Intubation is indicated if the pO_2 is <60 mmHg in adults or <80 mmHg in children (while on high-flow oxygen).
 - d. If the patient has signs of pulmonary insufficiency, use continuous positive-airway pressure (CPAP) if awake, or positive end-expiratory pressure (PEEP) if comatose. Indications include:
 - (1) A very high respiratory rate (50 breaths per minute)
 - (2) A $PaO_2 <60$ mmHg with an $FiO_2 <50\%$
 - (3) A $PaCO_2 >35$ mmHg
 - (4) A PaO_2 to FiO_2 ratio <300
 - e. Persistent hypoxemia may be due to aspiration of particulate matter that may require bronchoscopy.
4. Treat bronchospasm: aerosolized albuterol 0.5% solution, 2.5 mg in 2–3 mL normal saline every 20–30 minutes as needed
 5. Establish an IV of normal saline or lactated Ringer's (if not started in the field), and place patient on a cardiac monitor and pulse oximeter.
 6. Correct shock (if present) with fluid boluses; if this is unsuccessful, use an inotrope (eg, norepinephrine).
 7. Maintain cervical spine immobilization until cervical and first thoracic vertebra have been cleared by imaging.
 8. Decompress the stomach with a nasogastric tube.
 9. Place Foley catheter to monitor urine output.
 10. Conduct a thorough search for associated injuries.
 11. Assess the patient's temperature to exclude hypothermia (which is not uncommon and changes the therapeutic approach).
 - a. Use a rectal thermometer that is specially calibrated to record low temperatures; most clinical thermometers only read down to 95°F (35°C).
 - (1) Patients who have been submerged in icy cold water ($\leq 41^\circ\text{--}50^\circ\text{F}$ [$5^\circ\text{--}10^\circ\text{C}$]) for >40 minutes have survived with a good neurologic outcome.
 - (2) Cold water slows the metabolic rate and shunts blood to the brain, heart, and lungs (diving reflex).
 - b. Institute appropriate rewarming measures for hypothermia.
 - c. Extracorporeal membrane oxygenation (if available) is indicated for patients with severe hypothermia and/or hypoxia.
 - d. Continue resuscitation efforts until a near-normal core temperature (95°F [35°C]) is attained.
 12. Diagnostic evaluation
 - a. Cervical spine imaging should be done if there is any suspicion of possible cervical injury.
 - b. Arterial blood gases
 - c. Chest radiograph (50% of patients with abnormal radiographs will require intubation). Typically, one of three patterns is seen:
 - (1) Normal (although this does not necessarily mean there is no lung pathology)
 - (2) Perihilar pulmonary edema
 - (3) Generalized pulmonary edema

- d. CBC and urinalysis
 - e. Glucose, electrolytes, prothrombin time (INR)/partial thromboplastin time
 - f. Blood alcohol level and drug screen
 - g. ECG
13. Do not administer prophylactic antibiotics or steroids; neither has been shown to change the course of aspiration pneumonia in submersion-injury victims.

F. Predicting outcome

1. The most reliable predictors of outcome are the duration of submersion and resuscitation.
 - a. Good prognostic indicators
 - (1) Short submersion time
 - (2) Basic and/or advanced life support at the scene
 - (3) Favorable response to initial resuscitative efforts (return of spontaneous respiration)
 - (4) Alert on admission
 - (5) Older child (≥ 3 years old) or adult
 - (6) Water temperature 41°–50°F (5–10°C)
 - b. Bad prognostic indicators
 - (1) Submersion duration >25 minutes
 - (2) Cardiac arrest requiring >25 minutes advanced life support
 - (3) Ongoing CPR in the emergency department
 - (4) Fixed, dilated pupils in the emergency department
 - (5) pH <7.1
 - (6) Age <3 years
 - (7) Glasgow Coma Score <5 in the emergency department
2. Classification of submersion-injury victims based on the neurologic examination within 1 hour of rescue is also highly predictive of outcome.
 - a. Patients who are awake and alert almost always survive intact.
 - b. Patients who are blunted (obtunded to stuporous) but arousable and have a purposeful response to painful stimuli usually recover without neurologic sequelae.
 - c. Patients who are comatose and have an abnormal response to pain and abnormal respirations have a more variable outcome; although some survive intact if appropriate care is provided, many die or survive with anoxic encephalopathy. These patients were generally submerged for a prolonged period of time.
3. Pulmonary injury and hypoxia are the primary pathophysiologic determinants of outcome.

G. Disposition

1. Asymptomatic patients should be observed for 6 hours. If they remain asymptomatic, are oxygenating normally on room air, and have a normal chest radiograph, they may be discharged. Adequate follow-up should be done.
2. Hypoxemic and/or symptomatic patients should be hospitalized.

H. Postimmersion syndrome risk factors

1. Severe transient hypoxia
2. History of unconsciousness in the water
3. Presence of symptoms such as dyspnea, coughing, and tachypnea
4. Underlying cardiopulmonary disease

VII. OTHER ENVIRONMENTAL HAZARDS

A. Radiation injuries

1. Pathophysiology: radiation → ionization → formation of free radicals from water → breakage of DNA and RNA strands
2. The LD50 of whole-body ionizing radiation (the dose that will kill 50% of those exposed to it) is 350 rad (3.5 Gy).
3. The whole-body dose of ionizing radiation determines the timing of the onset of symptoms, severity of the illness, and organ system involved.
 - a. The higher the exposure, the earlier symptoms develop: <2 hours (>400 rads), >6 hours (<75 rads).
 - b. Organ systems with higher cell turnover rates are the most sensitive to radiation effects and will cause symptoms earlier and at lower doses than organ systems with lower cell turnover rates.
 - (1) The hematopoietic system is the most sensitive; radiation effects are detectable at 75 rads.
 - (2) The GI system is the next most sensitive.
 - (a) Mild effects (anorexia, nausea, vomiting) → 75–125 rads
 - (b) Moderate effects (nausea, vomiting, and diarrhea = acute radiation syndrome) → 100–200 rads
 - (c) Significant GI complications (massive fluid/plasma protein loss, gram-negative sepsis → >500 rads
 - (3) The CNS is the most resistant to radiation effects; exposure >5,000 rads is required to damage this system.
4. The best predictor of survival is the absolute lymphocyte count 48 hours after radiation exposure.
 - a. >1,200/mm³ = good prognosis
 - b. 300–1,200/mm³ = fair prognosis
 - c. <300/mm³ = poor prognosis
5. Most radiation accidents occur in an industrial setting. Most of the injuries are burns (usually the hands). Most fatalities are due to total body contamination secondary to occupational radiation devices (sealed sources, x-ray devices, or accelerators).
6. Radiologic weapons (ie, “dirty bombs”) are a growing concern for radiation injury.

B. Crush syndrome

1. Pathophysiology: compression of a body part (usually a limb) for at least 4 hours → release of skeletal muscle intracellular contents → rhabdomyolysis and electrolyte abnormalities → acute renal failure and cardiotoxicity
2. Clinical presentation
 - a. May look like a spinal cord injury but normal bladder function and intact anal sphincter tone exclude this.
 - b. Flaccid paralysis and sensory loss that is not related to nerve distribution
3. Fluid resuscitation
 - a. Should be started at the scene before extrication and maintained during extrication. Administer normal saline at 1.5 L/hr (to be continued en route to the hospital).

- b. On arrival in the emergency department, aggressive fluid therapy remains the primary focus of management until the blood pressure is stabilized and urine output is 1.5–2 mL/kg/hr.
- 4. Emergency fasciotomy should be done only as a “last resort” in the face of high intracompartmental pressures (as documented by direct manometry).

C. Volcanic eruptions

- 1. Mortality
 - a. Immediate: ash in the upper airways → suffocation
 - b. Delayed: ash (or other particulate matter) in the lungs → ARDS
- 2. Morbidity
 - a. Increased incidence of new-onset asthma
 - b. Prolonged inhalation of volcanic ash → silica pneumoconiosis

D. Blast injuries

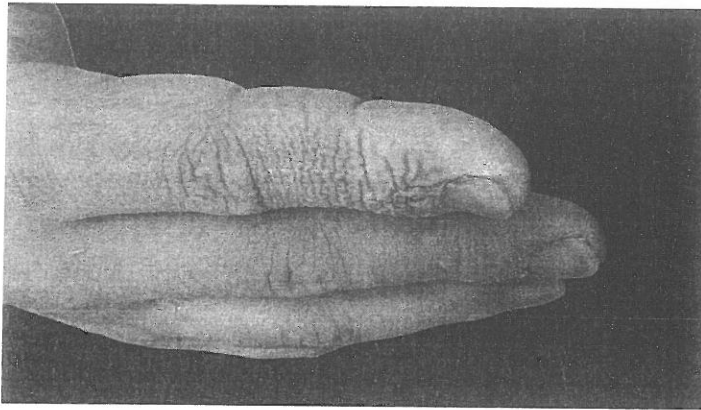
- 1. **Physical trauma from direct or indirect exposure to an explosion**
- 2. **Four classes of injury**
 - a. **Primary**
 - (1) Caused by blast overpressure or shock waves
 - (2) Ears most commonly injured, followed by lungs and GI tract
 - (3) External injuries often absent
 - b. **Secondary**
 - (1) Caused by objects propelled by the explosion
 - (2) Often penetrating trauma
 - (3) Most casualties caused by secondary injuries
 - c. **Tertiary**
 - (1) Displacement of air by the explosion throws victims against solid objects
 - (2) Often combination of blunt and penetrating trauma
 - (3) Persons who weigh less (eg, children) are at higher risk.
 - d. **Quaternary**
 - (1) Includes all other injuries: flash burns, crush injuries, respiratory injuries, etc
 - (2) Psychiatric injury is the most common quaternary injury.

ENVIRONMENTAL DISORDERS: PRACTICE CLINICAL SCENARIOS

Answers immediately follow the practice clinical scenarios.

Scenario A

Presentation: A middle-aged antique dealer presents complaining of hand discoloration and weakness. His symptoms started after using a rust and glass cleaner on some newly purchased glass sculptures and metal etchings. Laboratory studies are normal except for hypocalcemia.



Source: Dr. Watchorn (Wikimedia Commons: http://en.wikipedia.org/wiki/File:61569264_jamesheilman-224x2991.jpg)

What is the diagnosis?

Scenario B

Presentation: A 4-year-old boy bites a household electrical cord and receives burn care at a local urgent care that day. Three days later, his parents bring him to the local emergency department with profuse lip bleeding with arterial pulsations. ABCs are started, and an emergent plastic surgery consult is obtained.

What is the diagnosis?

Scenario C

Presentation: A grandfather flies a kite with his grandson on a warm summer day when a storm rolls in. The grandfather attempts to rapidly bring down the kite when he is hit by lightning. He thinks he tripped while bringing the kite down but only recalls waking up on a grassy knoll. He is asymptomatic other than Lichtenberg figures.



Source: United States Department of Health and Human Services (Wikimedia: http://commons.wikimedia.org/wiki/File:Rear_view_of_a_lightning-strike_survivor_displaying_Lichtenberg_figure_on_skin.png)

What is the diagnosis?

Scenario D

Presentation: A licensed California marijuana farmer brings in a dead snake. He states the snake bit him on the hand while he was trying to harvest his crop. The snake has wide red bands next to narrow black bands.

What is the diagnosis?

Scenario E

Presentation: An emergency physician from Ohio takes a morning flight into Denver after working all night. He and his wife drive immediately to a large ski resort a few hours outside of Denver. He begins to notice severe headache and nausea as he rides up the chairlift. His symptoms seem to improve as he descends the slope and recur as he rides back up the chairlift.

What is the diagnosis?

ANSWERS TO PRACTICE CLINICAL SCENARIOS

Scenario A

Diagnosis: hydrofluoric acid burn

Scenario B

Diagnosis: electrical lip burn

Management: Do not debride these wounds. Cleanse and apply a petroleum-based antibiotic ointment. Observe closely and refer to a plastic or oral surgeon for splinting and further care. Complications include delayed hemorrhage from the labial artery (10%–15% of patients) 3–14 days after injury, when the eschar separates, which is what occurred in this patient.

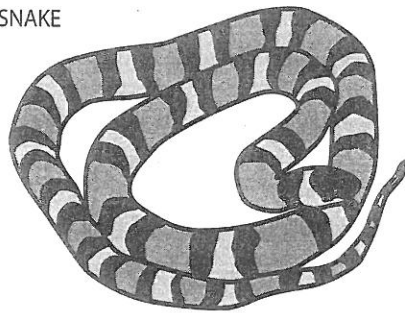
Scenario C

Diagnosis: lightning strike

Scenario D

Diagnosis: snake bite: "red on black, venom lack"

TEXAS CORAL
SNAKE



Red and Black
VENOM LACK



red | black | yellow | black | red

LOUISIANA MILK
SNAKE



Red and Yellow
KILL A FELLOW



yellow | red | black | red | yellow

Scenario E

Diagnosis: acute mountain sickness

NOTES

PSYCHOBEHAVIORAL DISORDERS

Clinical Approach to the Psychiatric Patient	861
Recognition	861
Triage	861
Safety	862
Medical Evaluation	862
The Psychiatric Interview	865
Treatment.....	866
Management of the Agitated Patient.....	866
Crisis Intervention.....	872
Psychopharmacology.....	874
Specific Behavioral Disorders	881
Psychosis	881
Depression	882
Suicide	884
Mania and Hypomania.....	887
Catatonia.....	888
Panic Attacks/Disorder	889
Somatoform Disorders	890
Insomnia and Sleep Disorders	892
Eating Disorders	893
Intoxication and Withdrawal.....	896
Delirium and Dementia.....	899
"Medical Mimickers" of Psychiatric Illness	901

PSYCHOBEHAVIORAL DISORDERS: SELF-ASSESSMENT QUESTIONS

1. Which of the following is not an adverse effect of antipsychotics?
 - (a) Hypotension
 - (b) Bradycardia
 - (c) Dystonic reactions
 - (d) Lowered seizure threshold
2. A depressed 20-year-old woman arrives in the emergency department complaining of headache, chest tightness, and palpitations. Examination reveals tachycardia, increased blood pressure, and diaphoresis. She mentions that she is taking a medication for depression but cannot recall its name. She states that she started to feel ill shortly after taking an OTC decongestant (cough suppressant) for her cold symptoms. These would be signs of a potentially lethal drug-drug interaction if the patient's antidepressant were a:
 - (a) Benzodiazepine
 - (b) SSRI
 - (c) Monoamine oxidase inhibitor
 - (d) Tricyclic antidepressant
3. The patient most likely to benefit from crisis intervention is the patient with:
 - (a) An acute grief reaction
 - (b) Delirium
 - (c) Agoraphobia
 - (d) Seasonal affective disorder
4. Which of the following statements regarding conversion reactions is inaccurate?
 - (a) They have a sudden onset and are often triggered by an emotionally charged event.
 - (b) The process is unconscious; the patient is not malingering.
 - (c) Symptoms attributed to conversion reactions are often later found to be due to an occult general medical disorder.
 - (d) The symptoms often involve involuntary muscle functions.
5. Which of the following medications has depression as an adverse effect?
 - (a) Propranolol
 - (b) Furosemide
 - (c) Sumatriptan
 - (d) Atorvastatin

6. Which of the following is not an identified risk factor for suicide?
 - (a) Recent surgery
 - (b) Drug and alcohol abuse
 - (c) Chronic pain
 - (d) Family history of suicide
7. Pharmacologic restraints are sometimes needed for a patient in the emergency department to protect the patient and others from harm. The agents of first choice for use in the emergency department are:
 - (a) Chlorpromazine and diazepam
 - (b) Haloperidol and lorazepam
 - (c) Thioridazine and midazolam
 - (d) Droperidol and clonazepam
8. Unlike the hallucinations that occur in patients with delirium due to an underlying medical condition, those occurring in patients with a psychosis (eg, schizophrenia) are usually:
 - (a) Tactile and painful
 - (b) Auditory and frightening
 - (c) Visual and colorful
 - (d) Olfactory and noxious
9. Which of the following is not considered a symptom of depression?
 - (a) Feelings of worthlessness or guilt
 - (b) Pressured speech
 - (c) Sleep disturbances
 - (d) Weight loss, weight gain, or change in appetite
10. Which of the following statements regarding antidepressants is accurate?
 - (a) Routine prescription of these agents to patients evaluated in the emergency department for depression is appropriate.
 - (b) SSRI antidepressants produce significant cardiotoxicity.
 - (c) SSRIs may influence other drug levels through cytochrome P450 pathways.
 - (d) Second-generation antidepressants (venlafaxine, duloxetine, mirtazapine) exert their mechanism of action through dopamine and norepinephrine effects.
11. Which of the following statements regarding panic attacks is incorrect?
 - (a) Symptoms include apprehension, shortness of breath, difficulty swallowing, chest tightness, and palpitations.
 - (b) Patients often believe they have a medical problem.
 - (c) Attacks are sudden in onset and last, on average, <30 minutes.
 - (d) Response to treatment is generally poor.

12. Which of the following complications occurs in anorexia nervosa only, rather than both anorexia nervosa and bulimia nervosa?
- (a) Over concern with weight or shape
 - (b) Pathologic fear of gaining weight
 - (c) Cardiac dysrhythmias
 - (d) Body weight loss <85% ideal body weight
13. Which of the following associations (psychotropic medications with complications) is inaccurate?
- (a) Olanzapine, lorazepam, phenelzine: dystonia
 - (b) Imipramine, doxepin, amitriptyline: QT prolongation and ventricular dysrhythmias
 - (c) Clozapine, bupropion, venlafaxine: tonic-clonic seizures
 - (d) Haloperidol, fluphenazine, risperidone: neuroleptic malignant syndrome
14. A 24-year-old woman with a history of bipolar disorder is brought to the emergency room by her family. Over the past day, she has been increasingly withdrawn and mute. She now refuses to talk to her family and resists the examiner's attempts to move her limbs. Vitals signs, laboratory studies, and head CT are all normal. The next step in diagnosis is to give:
- (a) Haloperidol 2.5 mg IV to treat psychosis
 - (b) Fluphenazine 5 mg IM to treat catatonia
 - (c) Ziprasidone 20 mg orally to treat psychosis
 - (d) Lorazepam 1 mg IM to treat catatonia
15. A 20-year-old woman presents with agitation, hallucinations, and abdominal pain. Her abdominal examination is benign, but she continues to be preoccupied with the pain. Her physician made a psychiatric referral and prescribed clonazepam for her anxiety, but her condition rapidly worsened and she became increasingly distressed by her abdominal pain before the psychiatric evaluation could be arranged. The most likely diagnosis is:
- (a) Manic psychosis
 - (b) Acute intermittent porphyria
 - (c) Somatoform pain disorder
 - (d) Hypocalcemia

ANSWERS

- | | | | | |
|------|------|------|-------|-------|
| 1. b | 4. d | 7. b | 10. c | 13. a |
| 2. c | 5. a | 8. b | 11. d | 14. d |
| 3. a | 6. a | 9. b | 12. d | 15. b |

Use the pre-chapter multiple choice question worksheet (page xx) to record and determine the percentage of correct answers for this chapter.

I. CLINICAL APPROACH TO THE PSYCHIATRIC PATIENT

A. Recognition

1. A psychiatric emergency arises when a patient poses a danger to others/self or is gravely disabled. The situation is complex and often characterized by intense symptoms, a sense of urgency, and perceived danger.
2. The patient's primary problem (depression, psychosis) may be identified by himself/herself, family or friends, or others who are aware of the patient's abnormal behavior.
3. Patients may present with physical symptoms that are manifestations of a psychiatric disorder, eg, a depressed patient may present with insomnia, weight loss, and somatic complaints.
4. Patients may present with psychiatric complaints that are caused or exacerbated by general medical syndromes or medications/substances (eg, psychosis due to corticosteroids or phencyclidine [PCP] ingestion).
5. Patients may present with a general medical problem or injury that was caused by an underlying psychiatric disorder (eg, dystonic reaction or a suicide attempt).
6. Patients may also present with a general medical condition that has been exacerbated by neglect or with symptoms that are masked or distorted by their psychiatric disorder.

B. Triage: determine the need for immediate treatment.

1. Psychiatric patients should be seen in accordance with guidelines established for all patients (by severity of illness, then "first come, first served"). Patients should be categorized as emergent, urgent, or nonurgent and seen in order of priority.
 - a. Emergent: ask yourself "Does this patient's condition pose a physical threat to himself or herself or others?" The answer is "yes" if:
 - (1) Vital signs are abnormal.
 - (2) A life-threatening illness or injury is present, even though the chief complaint is psychiatric (eg, the suicidal patient with a potentially lethal overdose or the combative patient who is hypoglycemic).
 - (3) The patient is suicidal, violent (or potentially violent), or unable to care for himself or herself.
 - b. Urgent: patient does not meet emergent criteria, and there is:
 - (1) Agitation
 - (2) Overwhelming anxiety
 - (3) Suicidal or homicidal ideation
 - (4) An inability on the part of the patient to explain the problem coherently
 - (5) Extremely concerned family/collaterals
 - (6) Intoxication
 - c. Nonurgent: patient requests psychiatric help but does not meet criteria for emergent or urgent treatment.
2. All patients need a medical (as well as psychiatric) evaluation before leaving the emergency department, including a complete set of vital signs and a mental status examination. People with psychiatric illness may have comorbid medical/surgical conditions that may be overlooked because staff has labeled the patient and/or chief complaint as psychiatric ("anchoring" phenomenon).

3. Patients with psychiatric problems must not be permitted to leave the emergency department without being evaluated as potentially harmful to self and/or others, incompetent to care for self, intoxicated, or psychotic and grossly disorganized.

C. Safety: determine the need for seclusion/restraints.

1. Attending to the safety of the patient, physician, and staff is a primary concern when managing a patient with agitation and/or thoughts of harm to self or others.
2. Many emergency departments have rooms designed with this population in mind. The room should be devoid of medical equipment that could be used to harm oneself (eg, IV tubing that could be used to strangle oneself) or others (eg, equipment that could be used as projectiles).
3. Patients who are reporting active suicidal ideation should be placed on a direct-observation status (eg, use of a one-on-one sitter).
4. Early warning signs indicating that a patient is a risk for violent behavior should be addressed immediately.
5. Maintain a safe distance between you and the patient, and allow for you and the patient to both have an easy egress from the room.
6. Keep in mind that the behavior of a patient with psychosis can be unpredictable, because the patient may be responding to thoughts (delusions) or perceptions (hallucinations) that are not based in reality.

D. Medical evaluation

1. **The goal is to recognize and distinguish general medical ("organic") from psychiatric ("functional") illness. If a general medical illness is identified or suspected, evaluate and treat appropriately before or while obtaining a psychiatric consult.**
 - a. **Profile of a general medical disorder presenting as a behavioral emergency**
 - (1) **Classic presentation: sudden or recent onset of a behavioral change in an older person without previously diagnosed psychiatric disorder**
 - (2) **The patient may retain insight (but not always).**
 - (3) **Specific findings**
 - (a) **Sudden onset or change in symptoms**
 - (b) **Recent diagnosis of a medical illness**
 - (c) **Recent change in prescribed or OTC medications**
 - (d) **Visual, tactile, or olfactory hallucinations are more common than auditory hallucinations.**
 - (4) **Abnormal cognitive function including:**
 - (a) **Altered level of consciousness (sedation or hyperarousal)**
 - (b) **Disorientation (confusion is usually present)**
 - (c) **Memory impairment**
 - (d) **Poor attention**
 - b. **Profile of a psychiatric disorder**
 - (1) **Past history of behavioral problems in a younger patient**
 - (2) **The patient lacks insight (but not always).**
 - (3) **Specific findings**
 - (a) **Auditory hallucinations are more common than other hallucinations.**
 - (b) **Normal vital signs, physical examination, and laboratory studies**

- (c) Disordered or illogical thinking, especially in the face of normal cognitive function. The patient should be oriented and can complete routine cognitive tasks (eg, spelling "world" backward).
- c. Specific medical illnesses presenting as psychiatric emergencies
 - (1) Common "behavioral" presentations
 - (a) Adverse effects of medications
 - (b) Hyponatremia
 - (c) Hypoglycemia or hyperglycemia
 - (d) Alcohol intoxication or withdrawal
 - (e) Illicit drug intoxication (eg, cocaine, bath salts, LSD, phencyclidine [PCP])
 - (f) Drug withdrawal
 - (g) Urinary tract infection leading to delirium
 - (h) Pneumonia leading to delirium
 - (i) COPD leading to hypoxia or hypercapnia
 - (j) Liver disease, which may be accompanied by hyperammonemia
 - (k) Chronic renal disease
 - (l) Hypothyroidism or hyperthyroidism
 - (2) Less common but potentially life-threatening "behavioral" presentations
 - (a) Subarachnoid or intracranial hemorrhage
 - (b) Encephalitis
 - (c) Metastatic carcinoma or intracranial tumor
 - (d) Malignant hypertension/hypertensive encephalopathy
 - (e) Hypocalcemia/hypercalcemia
 - (f) Steroid-induced psychosis
 - (g) Autoimmune diseases (eg, systemic lupus erythematosus)
- 2. Medical history
 - a. All history must be corroborated.
 - (1) Compare the patient's version with that of family, friends, and other sources (eg, police, paramedics).
 - (2) Obtain old medical and psychiatric records.
 - (3) Contact current or recent primary care physician.
 - b. Include medications, especially pain relievers, sedatives, and psychiatric medications. (Remember that many OTC and prescription drugs, as well as herbal supplements, have psychiatric adverse effects.)
 - c. Ask about drug and alcohol use (often overlooked in the professional, executive, "nice person," elderly, and children!)
- 3. Physical examination
 - a. If the patient is unable to cooperate, physical or chemical restraints may be required before examination.
 - b. If the patient refuses to cooperate, procedures for involuntary commitment and/or emergency assessment should be followed. Do not permit the patient to leave the emergency department before adequate medical and psychiatric assessments have been completed.
 - c. Patients must be disrobed and gowned, but first conduct search for possible weapons.

- (1) Proactively seek assistance from hospital security personnel and/or police.
- (2) If the patient has a weapon, security personnel are required; they must disarm him or her before any further evaluation or treatment can be safely conducted.
- d. Make sure vital signs have been taken and recorded (abnormalities suggest a medical problem).
- e. Neurologic examination: note particularly any signs of nonpsychiatric illness, eg, dysarthria, aphasia, dyspraxia, ataxia, dyskinesias, tremor, nystagmus, incontinence, paresis, paresthesias, and lateralizing neurologic signs.
- f. Documentation should include vital signs as well as signs of:
 - (1) Trauma or exposure-related injuries
 - (2) Previous suicide attempts
 - (3) Thyroid, liver, or renal disease
 - (4) Toxidromes (anticholinergics, sympathomimetics, opioids, etc)
 - (5) Withdrawal syndromes (opioids, benzodiazepines, alcohol, etc)
4. Mental status examination
 - a. Level of consciousness: alert/somnolent/stuporous, fluctuating, or stable
 - b. General appearance
 - (1) Activity level: agitation, hyper-, normo-, hypomotor
 - (2) Grooming and hygiene: appropriate, inappropriate, disheveled
 - (3) Movements: tremor, tics, stereotypes, chorea, dyskinesias
 - c. Orientation
 - (1) Time, place, person, situation
 - (2) Orientation to time is most sensitive: ask about day, date, month, season, and year.
 - d. Memory
 - (1) Immediate, short- and long-term
 - (2) Give the patient three words and ask him or her to repeat them (immediate recall).
 - (3) Ask the patient to repeat the words again after 3 minutes (short-term recall).
 - (4) Long-term recall can be inferred in the course of history-taking.
 - e. Mood and affect
 - (1) Mood is self-reported ("sad," "angry"), whereas affect is observed (flat, irritable, tearful).
 - (2) Affect ranges from flat to labile and can be described as mood-congruent or mood-incongruent (eg, inappropriate laughter).
 - f. Speech: rate, rhythm, fluency
 - g. Thought process: may be described as linear and logical, circumstantial, tangential, derailed, loose associations, thought blocking, illogical
 - h. Thought content
 - (1) Delusions ("fixed false beliefs")
 - (a) Paranoid, persecutory, grandiose, self-deprecatory, erotic
 - (b) Mood congruent/incongruent
 - (c) Bizarre (impossible in real life) versus nonbizarre
 - (2) Suicidal or homicidal ideation
 - (3) Depressive cognitions: hopelessness, worthlessness, inappropriate guilt

- i. Perceptual abnormalities
 - (1) Hallucinations: visual, tactile, auditory, olfactory
 - (2) Illusions: misperceptions of visual stimuli
 - (3) Dissociative phenomena: flashbacks, déjà vu, depersonalization
- j. Cognitive function: ask the patient to spell a word backward (such as "world" or "earth") and subtract 7 from 100 serially (93, 86, 79, etc).
- k. Judgment and insight: can usually be inferred in the course of history-taking
- 5. Screening diagnostic studies are not required to medically clear all patients with psychiatric complaints. The history, physical examination, and clinical judgment should prompt appropriate diagnostic evaluation.
 - a. "Medical clearance" is a controversial term used to communicate that the patient is stable for transfer to a psychiatric facility where medical resources may not be readily available.
 - b. The history and medical examination may indicate the need for diagnostic evaluation, including electrolytes, BUN, creatinine, urinalysis, CBC, glucose, ECG, and chest radiograph.
 - c. Tests such as an ethanol level or urine drug screen may aid psychiatric consultants in making an appropriate diagnosis and referral.
 - d. Drug levels, including lithium, valproic acid, and carbamazepine, are useful in determining medication compliance. Trough levels are preferred for therapeutic monitoring.
 - e. Be suspicious of suicidal patients who may not disclose an ingestion, and consider ordering acetaminophen and salicylate levels; these ingestions are best discovered early.

E. The psychiatric interview

- 1. General approach
 - a. Psychiatric conditions, like all medical problems, are biopsychosocial disorders. Evaluation should focus on signs and symptoms (onset, severity, and progression), the patient's physical and behavioral coping, and also the availability of therapeutic and/or supportive resources.
 - b. Stages of the interview
 - (1) Begin by asking open-ended questions and be silent afterward; the patient will fill in the silence.
 - (2) Progress to structured, directive questioning to explore symptoms and circumstances.
 - (3) Finish with a psychiatric "review of systems" to continue to consider or exclude covert, comorbid syndromes.
 - (4) All history must be corroborated. In addition to the sources listed under the medical evaluation, contact with the patient's current treating psychiatrist and psychotherapist is essential to rapid, accurate diagnosis and treatment planning.
 - (5) Collateral information is essential, particularly when planning to discharge a patient with safety concerns. It is considered standard of care.
 - c. Pay attention to your emotional response to the patient.
 - (1) The interviewer's emotional response will often mirror the patient's mood and thus is a valuable diagnostic instrument.
 - (2) Maintain a calm demeanor and nonjudgmental approach with even difficult patients. Arguing with an agitated patient may exacerbate the situation; a better approach is to agree with the patient or "agree to disagree."

2. The goals of the interview are to determine the:
 - a. Nature of the problem: "What is wrong?" What is the patient upset or worried about, and how is he or she having difficulty functioning?
 - b. Precipitant: "Why now?" What change in symptoms or circumstances has led to the current emergency department visit, and how did the patient come to the emergency department today?
 - c. Progression, including course and duration: How long has the problem been going on, how has its nature and severity changed, has there been any prior treatment, and if so, to what effect?
 - d. Patient's expectations: What is he or she (or the patient's family) hoping to have done at this time?
 - e. Disposition: Is an appropriate plan medical admission, psychiatric admission, or outpatient psychiatric and/or medical follow-up?
3. The interview: in addition to asking questions to determine the patient's mental/emotional stability, ask specifically about:
 - a. Suicidal or homicidal ideation or threats
 - b. Availability of firearms
 - c. Hallucinations, delusions
 - d. Drug, alcohol, and prescription medication abuse
 - e. Current (or past) physical or sexual abuse
 - f. Any inconsistencies between the histories given by the patient and collaterals, or between the history given and your examination findings.
4. Determining disposition
 - a. Inpatient versus outpatient management is determined by whether the patient presents immediate danger to himself/herself or others, whether the patient is able to care for himself/herself, and what social supports are available at home.
 - b. At the end of the interview, discuss your understanding of the psychiatric problem and any medical/surgical problems, and explain treatment recommendations.
 - c. If immediate psychiatric hospitalization is indicated but unavailable (on site or by transfer), some hospitals will admit the patient to a medical service with appropriate supervision ("sitter") and psychiatric consultation, while other hospitals will have the patient "board" in the emergency department.

II. TREATMENT

A. Management of the agitated patient

1. Agitation may reflect a number of underlying problems.
 - a. Neurologic problem, such as head injury or postictal state
 - b. Delirium
 - c. Intoxication or withdrawal from substances
 - d. Psychosis or mania
 - e. Dementia
 - f. Personality traits that result in anger outbursts and tendency to be violent in the absence of the above

2. Recognize early warning signs of agitation.
 - a. Increased motor or verbal activity, eg, pacing in the room, clenched fists, a change in the volume or tone of voice
 - b. An abrupt change in behavior during the interview
 - c. Abnormal behavior exhibited in addition to signs of illness or injury
 - d. Patient creates anxiety or discomfort in emergency department staff
 - e. Patient states fear of losing control
 - f. Violence or threatened violence
3. Address signs of agitation early
 - a. Recognize that agitation is a continuum and may respond to various interventions.
 - b. Offering food, nicotine replacement, or an explanation for a long wait may go a long way toward calming an irritated patient.
 - c. Offering pharmacologic interventions early (eg, an atypical antipsychotic to a schizophrenic patient) may prevent the need for seclusion or restraints later during the course of the emergency department visit
 - d. Seclusion or restraints are necessary if a patient's uncontrolled behavior is potentially dangerous to himself/herself, other patients, or emergency department personnel.
4. Verbal de-escalation
 - a. The physician's tone of voice, comments to the patient, and body language can be an intervention that may de-escalate the patient and decrease the risk of violence. Approach the patient as if you expect him or her to demonstrate self-control and become cooperative, and keep a distance of at least 8 feet.
 - b. Place the patient in a quiet room or area, and remove family or friends who are not being helpful.
 - c. Speak to an agitated patient in a calm tone of voice and with direct statements. Introduce yourself, reassure the patient, orient him or her to the surroundings, and explain what you are going to do.
 - d. Aim to understand why the patient is angry. Validate these concerns, and let the patient know what to expect. For example, "I understand that you thought you would receive a prescription by coming to the emergency room. I would be upset, too, if I spent two hours waiting for a prescription and then was told that I would not get it. However, it is our policy not to give prescriptions for that medication."
 - e. Do not argue with the patient or raise your voice in reaction to the patient. Yelling will make the patient more defensive, and the agitation will increase. Patients who remain agitated or become threatening should be informed that such behavior is not allowed, is scaring others (you, staff, other patients), and that restraints (either physical or chemical) may be required to control the behavior.
 - f. The physician's body language should communicate confidence and calmness. Arms should be relaxed at the sides. Arms crossed in front of you can communicate that you feel afraid or closed off, whereas hands on the hips communicate that you are controlling and may be threatening.
 - g. Always keep clear a lane of egress from the room when evaluating a psychiatric patient with the potential for violence and/or inappropriate behavior.
 - h. Verbal de-escalation is particularly likely to be successful in a patient who is cognitively intact.
5. Pharmacologic management of agitation

- a. Use of medications to treat agitation is sometimes called "chemical restraint." Medications can be useful in obtaining behavioral control and should be considered once the initial evaluation has been completed or if the patient becomes agitated.

(1) Indications

- (a) Agitation that would otherwise interfere with safe medical management
 - (b) Treatment of the underlying cause of agitation (eg, giving an antipsychotic medication to a psychotic patient will decrease his or her agitation and also treat the psychosis)
 - (c) If the patient requests a medication that was previously effective for safety
- (2) Goal: a calm patient in behavioral control (not a patient who is too sedated to interview, although sleep may result from these medications)

(3) Relative contraindications

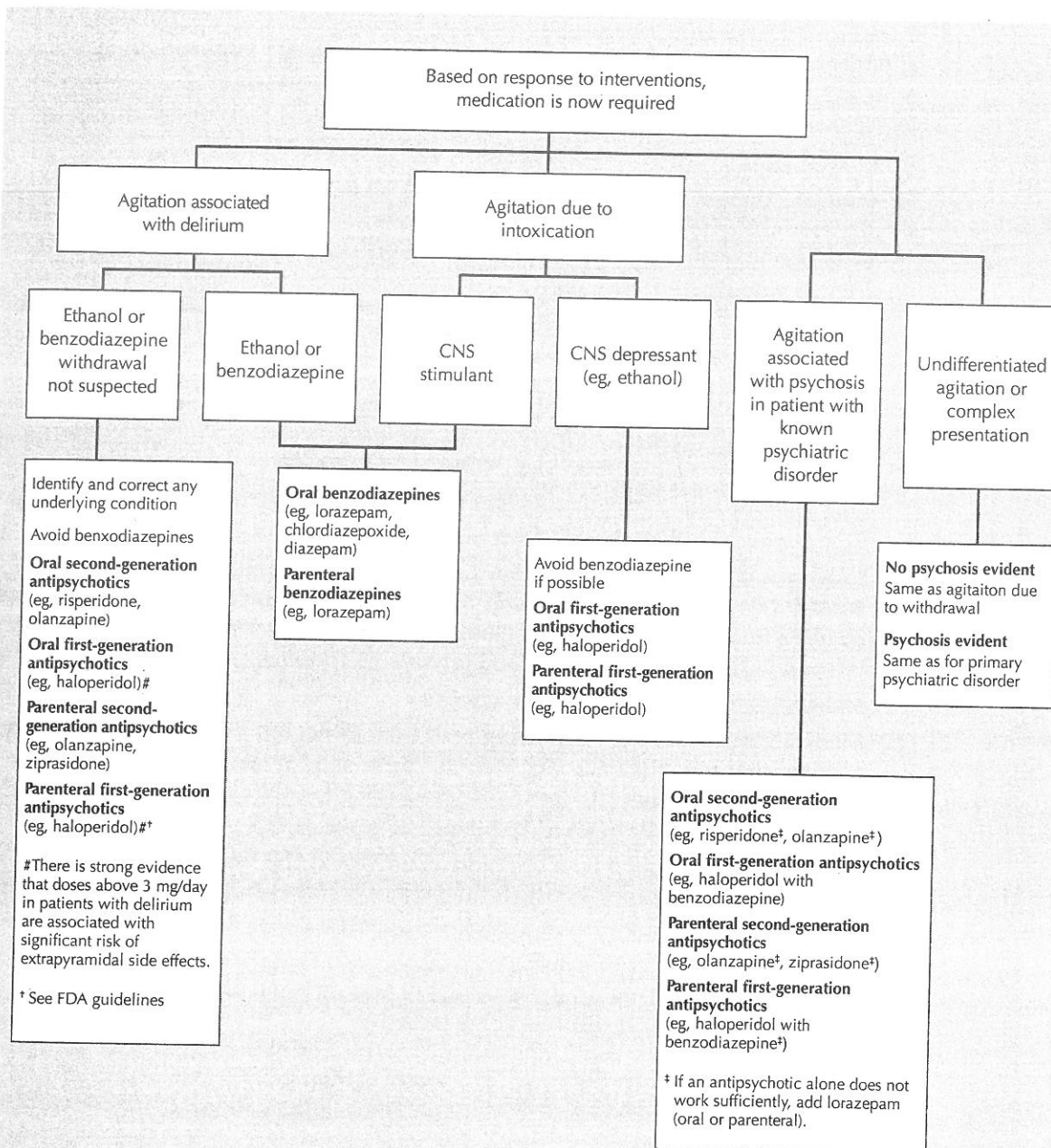
- (a) Incomplete assessment
 - i. Medication may obscure diagnostic signs and symptoms.
 - ii. Overmedication may delay completion of evaluation by several hours.
- (b) Unknown dosage of preexisting medication or overdose
- (c) For patients with dementia or other cognitive disorders, sedating medication (especially benzodiazepines) may cause disinhibition, potentially worsening agitation or confusion.

(4) Protocols

- (a) Administration of the oral concentrate form is preferred if the patient is willing because:
 - i. Consent is thereby implied.
 - ii. Therapeutic alliance with the patient is furthered.
 - iii. Absorption and efficacy are almost as rapid as with IM dosing.
- (b) "Rapid tranquilization" is well researched as safe and effective when using haloperidol, lorazepam, or both. This combination is considered by many to be the safest and most effective protocol.

b. Classes of medication

- (1) Medications should be chosen to target the underlying cause of the agitation.



Protocol for Treatment of Agitation

Source: Wilson MP, Pepper D, Currier GW, Holloman GH Jr, Feifel D. The psychopharmacology of agitation: consensus statement of the American Association for Emergency Psychiatry Project BETA Psychopharmacology Workgroup. *Western J Emerg Med*. Vol XIII, No. 1; Feb 2012:31.

- (2) If a patient is violent or at imminent risk of violent behavior, then medications may be given IM against the patient's consent. Hospital security should be present to restrain the patient while the medication is administered to decrease risk of staff or patient injury.
- (3) Haloperidol and lorazepam are frequently considered agents of choice and may be used orally, IM, or IV (alone or in combination). They may be given together in the same syringe. In the absence of contraindications, combined dosing tends

to reduce adverse effects as well as promote a synergistic drug effect. Lorazepam effectively prevents dystonia, while haloperidol permits the use of lower doses of lorazepam, thereby minimizing lorazepam's dose-dependent adverse effects (excessive sedation, respiratory depression).

(4) First-generation (typical) antipsychotics

- (a) Haloperidol 2–5 mg orally, IM, or IV every 30 minutes as needed up to 40 mg total for adults (0.5–1.0 mg every 30 minutes as needed in the elderly or children). Major complication is acute dystonias (may be prevented or treated with diphenhydramine 25–50 mg or benztropine 1 mg). Haloperidol is relatively contraindicated in patients with parkinsonism because of its potent D2 blockade.
- (b) Chlorpromazine 25–50 mg orally or IM every 30 minutes as needed. Hypotension may result with use of low-potency antipsychotics, particularly with concomitant use of a benzodiazepine.
- (c) Droperidol 5 mg orally, IM, or IV every 30 minutes as needed. The chief advantage is more rapid onset of action after IM administration (5 minutes, as opposed to 20–30 minutes with IM haloperidol). Duration of sedative effect is relatively short, 2–4 hours (versus a half-life of 10–19 hours for haloperidol). Torsades de pointes is a potentially serious complication of droperidol and has resulted in its elimination from many hospital formularies. Measure the QT interval before administration, and provide continuous cardiac monitoring.

(5) Second-generation (atypical) antipsychotics

- (a) Medications in this class are being used increasingly in psychiatric emergency services, because they are less likely to cause extrapyramidal symptoms than older antipsychotics.
- (b) Olanzapine 5–10 mg orally or IM every 30 minutes, up to a total dose of 30 mg. It is also available in a rapidly dissolving wafer that can be useful in emergency situations in which the patient is somewhat cooperative. Do not give IM olanzapine along with IM lorazepam because of the risk of oversedation and hypotension.
- (c) Ziprasidone 10–20 mg orally or IM every hour, up to a total dose of 40 mg; may cause QT prolongation in some patients.
- (d) Risperidone does not have an immediate-acting IM formulation, but its oral preparation may still be helpful in treating agitation.

(6) Benzodiazepines

- (a) Lorazepam 1–2 mg orally, IM, or IV every 30 minutes (0.25–0.5 mg every 30 minutes as needed in the elderly or children). Lorazepam is the only benzodiazepine with reliable and rapid IM efficacy.
- (b) Duration of action is intermediate (~6 hours).
- (c) Elimination is by hepatic conjugation and is not subject to cytochrome P450 interactions; no active metabolites are formed.
- (d) Respiratory depression may occur, particularly in COPD, preexisting sedative-hypnotic ingestion, or in IV use. (IV administration requires availability of intubation and respiratory support.)
- (e) Disinhibition and agitation may occasionally occur.
- (f) In cases of substance abuse, particularly cocaine intoxication, benzodiazepines alone are preferable choices for chemical restraint.

6. Seclusion

- a. The Centers for Medicare and Medicaid Services mandate detailed documentation of indications and procedures when restraints are used: "Seclusion or restraint can only be used in emergency situations... only when less restrictive measures have been found to be ineffective to protect the patient or others from harm." Restraints may be ordered only for limited periods, and continuous face-to-face or audio-video monitoring must be implemented. Transfers to other facilities are also subject to documentation of "least restrictive available" and "face-to-face monitoring" by trained staff.
 - b. A seclusion room is designed and located to provide:
 - (1) Safety: no potential weapons, minimal furnishings
 - (2) Reduced stimulation: soundproofed, dimly lit, private
 - (3) Continuous observation: by audio-video or in direct view of staff
 - (4) Security staff and resuscitation area nearby
 - (5) Clearly controlled entrance and exit
 - c. Vital signs and triage should be done before leaving a patient in the seclusion room.
7. Physical restraints (frequently required for violent or severely agitated psychotic, delirious/demented, or intoxicated patients)
- a. Advantages
 - (1) Minimal adverse effects
 - (2) Immediately reversible
 - (3) Clearly visible reminders to staff and patient
 - (4) Permits search for weapons and drugs (never, however, a primary indication)
 - (5) Legally, it is appropriate medical treatment that is clearly established (Youngberg v. Romeo).
 - b. Indications
 - (1) Patient is a danger to self or others.
 - (2) Patient is unable to cooperate with assessment of a life-threatening condition.
 - (3) Patient requests restraints for safety (rare).
 - c. Contraindications
 - (1) Unstable orthopedic injury
 - (2) Unstable cardiovascular status
 - (3) Use as punishment or solely for staff convenience
 - d. Protocol should be clearly established, in written form, and institution-specific, with all emergency department personnel well-trained.
 - (1) Call for help immediately; the presence of a "show of force" is frequently very helpful in de-escalating such situations.
 - (2) A team approach is required, with a leader and five helpers (one takes the head and each of the others an extremity).
 - (3) Use leather (not cloth) restraints on all four extremities.
 - (4) Restrain the patient so he or she can be examined and rolled onto his or her side quickly, if needed, to protect the airway. The patient should be in the prone position.
 - (5) Tell the patient why restraints were applied.

- (6) Search for weapons and drugs.
- (7) Perform a physical examination.
- (8) Monitor continuously, face-to-face, or by audio-video.
- (9) Frequently (every 15 minutes or less) reassess the patient for safety and the need for continued physical restraints.
- (10) Document in the chart why restraints were required (to protect the patient and/or staff from harm) and the frequency and results of reassessments. Have an institutional policy and follow it.
- (11) Remove restraints only when the patient's behavior improves and he or she is no longer a threat. Don't bargain for removal. If a patient is calm and in behavioral control, do not keep him or her in restraints longer than needed as a form of punishment or prophylaxis against future episodes of agitation.

B. Crisis intervention

1. A patient with or without a psychiatric illness may present to the emergency department "in crisis," eg, after a trauma or in bereavement. The emergency physician can follow guidelines for crisis intervention to help the patient manage the crisis.
2. Trauma may result in a severe psychological reaction. When the traumatic event is so severe or occurs so suddenly that patients are unable to master the resulting emotions, the patient experiences a crisis. Crisis intervention involves understanding the patient's view and assisting patients to change their perception of the problem in such a way that they are no longer overwhelmed by their own reaction.
 - a. Goals
 - (1) Stabilize the patient's emotional and behavioral reaction.
 - (2) Prevent psychological sequelae and screen for pathologic reactions.
 - (3) Assist the patient in developing mastery of the traumatic situation, thereby creating growth, maturation, and a long-term improvement in coping skills.
 - b. Prerequisites
 - (1) The traumatic event must be identified and must have ended.
 - (2) The patient must have sufficient self-reliance and social support.
 - c. Patients most likely to benefit are those with a prior history of adequate functioning.
 - d. Technique
 - (1) Develop rapport. Be supportive of the patient but avoid glib reassurance; mobilize hope by communicating an expectation for improvement. Reassure the patient that it is good that he or she is seeking help and that the catastrophe has indeed ended.
 - (2) Let the patient talk about his or her feelings; be a sympathetic, nonjudgmental, active listener.
 - (3) Help the patient to reassess the situation, survey critical issues, and develop a solution.
 - (4) Activate natural support systems in family and community.
 - (a) If possible, access community resources (eg, government agencies, caseworker, minister, responsible family member, etc) before discharging the patient from your care.
 - (b) Medical/surgical issues (eg, chronic pain) must be addressed.

- e. Warning signs of poor coping include dissociation, reexperiencing the traumatic event, hyperarousal (including hypervigilance or severe sleep disturbance), or avoidance of reminders of the trauma. These symptoms may suggest the need for referral to a counselor or psychiatrist.
 - f. Debriefing (ie, discussion of the event in detail, often in a group of individuals who had witnessed the same trauma) was previously recommended as a method to help individuals process a traumatic event. However, recent evidence suggests that group debriefing may increase the risk of developing an acute stress reaction or post-traumatic stress disorder and should be avoided.
3. Bereavement ("acute grief reaction")
- a. Etiology: awareness of a sudden loss, eg, sudden infant death syndrome, accidental death or suicide of a family member or close friend, spousal abandonment, job loss, etc
 - b. Clinical stages of grief and mourning have been described, some or all of which the patient may evidence.
 - (1) Shock and denial (may be accompanied by screaming/collapse)
 - (2) Anger
 - (3) Bargaining
 - (4) Depression (feeling of powerlessness over the event)
 - (5) Acceptance (in which loss and its consequences are constructively integrated into the patient's psyche and life)
 - c. Exclude "pathological grief reaction" ie, depressive mood disorder.
 - (1) Features characteristic of major depression (eg, pervasive sadness with insomnia, anorexia, agitation, impaired concentration, etc) may be found during the course of normal bereavement.
 - (2) Some symptoms are abnormal (even in severe bereavement) and warrant a diagnosis of "depressive disorder" and psychiatric treatment.
 - (a) Persistence of significant symptoms beyond 2 months
 - (b) Suicidal ideation (other than wishing to be with the deceased)
 - (c) Morbid preoccupation with worthlessness
 - (d) Marked slowing of thought and movement ("psychomotor retardation")
 - (e) Prolonged and severe debilitation
 - (f) Hallucinations other than hearing the voice of, or transiently seeing the deceased (these experiences are common—and normal—in survivors)
 - (g) Substance abuse or an increase in use of alcohol, pain medications, sedatives
 - d. Treatment
 - (1) Help patients identify the loss and encourage them to express their feelings about it; defuse blame, suspicion, and guilt.
 - (2) The severity of the grief reaction is directly related to the suddenness and importance of the loss; signs of severe depression, psychosis, or other inappropriate response warrant psychiatric referral.
 - (3) Most people will not require counseling; however, referral information should be provided, because some individuals may decide to use it later on.

C. Psychopharmacology

1. Antianxiety agents

a. Benzodiazepines

- (1) Useful for short-term management of anxious, agitated patients in crisis and for initial treatment of patients with panic disorder. They are also used to treat seizures, muscle tension, alcohol withdrawal, and catatonia.
- (2) When used in conjunction with antipsychotic agents, they have synergistic effects.
- (3) Sedation with low doses reduces anxiety; occasionally, disinhibition ("paradoxical excitation") occurs.
- (4) Lorazepam
 - (a) 0.5–2 mg may be repeated every 30–60 minutes as needed.
 - (b) Onset of action varies with route of administration; 20–40 minutes when administered orally or IM and <5 minutes when administered IV.
 - (c) Duration of action is 6–8 hours.
- (5) Chlordiazepoxide 25–100 mg is sometimes given for alcohol withdrawal, particularly in a patient without liver dysfunction. Its long half-life provides a self-taper.
- (6) Diazepam 5–10 mg can be administered orally or IM. Diazepam is highly distributed in fat, and will (along with several active metabolites) accumulate after repeated administration.
- (7) Adverse effects
 - (a) Dose-dependent sedation, impairment of motor coordination and reaction time
 - (b) Impairment of consolidation of short-term memory (learning); at higher doses, ataxia/dysarthria and amnesia ("blackouts") may occur.
 - (c) Respiratory depression is found at higher dosages but is essentially benign in oral and IM use for healthy individuals who are not taking other respiratory depressants. IV use, comorbid COPD (or other respiratory compromise), and interactions with other medications/substances can cause lethal apnea. Therapeutic use under these circumstances requires caution, less aggressive dosing, and the immediate availability of intubation and ventilatory support.
- (8) Tolerance may develop over time.
- (9) A withdrawal syndrome may occur with insomnia, agitation, increased vital signs, and withdrawal seizures.
- (10) These drugs have abuse potential and can be addictive. Diazepam and alprazolam are well-known offenders.
- (11) Benzodiazepines are very safe in oral and IM use.
 - (a) Lorazepam in particular has minimal cardiovascular effects, no active metabolites, and does not inhibit/induce cytochrome P450 isoenzyme catabolism of other substrates.
 - (b) Although these drugs are frequently ingested in suicide attempts, a fatality is rare unless they are taken in combination with alcohol or other drugs.

b. Buspirone

- (1) A nonbenzodiazepine anxiolytic that is not subject to withdrawal or addiction
- (2) Requires 2–3 weeks for efficacy and therefore is not indicated in the emergency department

- c. Antihistamines
 - (1) Diphenhydramine and hydroxyzine can be useful as mild oral antianxiety agents.
 - (2) Dosage is 25–50 mg every 4 hours orally or IM (may also be given IV).
 - (3) If a hypnotic is indicated for brief outpatient use after emergency department discharge, these agents are preferred because of their low incidence of adverse effects and safety.
- 2. First-generation antipsychotic agents
 - a. First-generation ("typical") antipsychotics have largely been replaced by second-generation ("atypical") antipsychotics for routine control of psychiatric symptoms. However, the first-generation agents continue to be used in some patients, including some who need long-acting injectable formulations. Antipsychotic medications are also called neuroleptics.

Table 35: Characteristics of First-Generation (Typical) Neuroleptic Agents

Potency	Dystonia	Drug	Equivalent Dose (mg)	Anticholinergic	Sedation
high	high			low	low
↑	↑	Haloperidol	2	↑	↑
		Droperidol	2		
↓	↓	Fluphenazine	2	↓	↓
		Perphenazine	10		
low	low	Chlorpromazine	100	high	high

- b. Mechanism of action is dopaminergic receptor blockade in the mesolimbic area of the CNS. This may result in reduced hallucinations, delusions, anxiety, impulsivity, and aggression.
- c. All antipsychotics cause some orthostatic hypotension and reflex tachycardia; these adverse effects (which are due to α -adrenergic blockade) are more common and severe with low-potency neuroleptics (chlorpromazine).
- d. The seizure threshold is lowered with use of antipsychotics.
- e. Haloperidol is the neuroleptic agent of choice in many emergency departments; it reduces agitation, has benign cardiovascular (and minimal anticholinergic) effects, is not very sedating, and has a rapid onset.
- 3. Second-generation antipsychotic agents
 - a. Characteristics
 - (1) Enhanced mechanism of action: postsynaptic antagonism of both serotonin and dopamine receptors
 - (2) Less likely to cause extrapyramidal symptoms but more likely to produce metabolic syndrome than first-generation agents
 - (3) Also used to treat bipolar disorder and to augment treatment of depression
 - b. Clozapine
 - (1) Very low risk of extrapyramidal symptoms, increased risk of seizures
 - (2) Risk of agranulocytosis requires weekly CBC monitoring. Its use is limited to psychotic disorders unresponsive to standard agents or to patients with severe extrapyramidal symptoms. It should never be started in the emergency department.

- c. Risperidone, paliperidone, ziprasidone, olanzapine, quetiapine, and aripiprazole are other second-generation antipsychotics. These agents may cause sedation, weight gain, hyperlipidemia, and hyperglycemia.
- 4. Adverse effects of antipsychotic agents
 - a. **Extrapyramidal symptoms: occur more often with high-potency neuroleptics but can also occur with second-generation agents**
 - (1) **Dystonias**
 - (a) Painful clonus of voluntary muscles; they usually involve muscles of the face, neck, and tongue, but any muscle group can be affected.
 - (b) Laryngospasm occurs rarely and can be life threatening.
 - (c) Occulogyric crisis involves involuntary eye movements, usually in the upward direction.
 - (d) Torticollis is a painful contraction of the sternocleidomastoid muscle.
 - (e) These reactions generally occur within the first month of treatment (or after dose increases).
 - (f) Treatment is with anticholinergic agents, followed by a maintenance prescription of anticholinergics prescribed twice daily to prevent recurrence.
 - i. Benztropine 1–2 mg orally, IM, or IV
 - ii. Diphenhydramine 25–50 mg orally, IM, or IV
 - (2) **Parkinsonism**
 - (a) One or more of the following: shuffling gait, bradykinesia or akinesia, resting tremor, cogwheel rigidity, masked facies, drooling
 - (b) Dosage reduction and/or anticholinergic agents are usually effective.
 - (3) **Akathisia**
 - (a) An unpleasant and disturbing feeling of restlessness with an almost uncontrollable desire to move; easily mistaken for agitation
 - (b) Reactions begin several days to weeks after start of therapy and are worsened by increases in dosage.
 - (c) **Treatment**
 - i. Propranolol 10–20 mg orally is the drug of choice for managing antipsychotic medication–induced acute and chronic akathisia, and relief is usually seen within 20–40 minutes; if not, the dose may be repeated. Ongoing treatment is necessary to prevent recurrence.
 - ii. For acute akathisia in the emergency department, treat with anticholinergics (eg, diphenhydramine) and/or lorazepam IV or IM.
 - (4) **Tardive dyskinesia**
 - (a) A disorder of abnormal, involuntary movements that develops after a period of antipsychotic use
 - (b) Oral-buccal muscle involvement is common and characterized by lip smacking and tongue protrusion. Limb and truncal muscles can also be involved.
 - (c) The patient can voluntarily suppress the movements temporarily.
 - (d) There is no definitive treatment. The offending agent should be discontinued. This should be coordinated with the treating psychiatrist to prevent recurrence of psychosis. Discontinuation of the antipsychotic may also lead to a transient increase in the movement severity but a greater chance of improvement over time.

- b. Anticholinergic effects (occur more often with low-potency neuroleptics)
 - (1) May be central (sedation, agitation, delirium/psychosis) and/or peripheral (eg, dry mucous membranes, flushed skin, mydriasis, tachycardia, urinary retention)
 - (2) Treatment is supportive, and the offending agent should be discontinued. Use of physostigmine should be avoided.
- c. Cardiovascular effects
 - (1) The major cardiovascular adverse effects (seen more often with low-potency neuroleptics) are hypotension (usually managed with IV fluids) and tachycardia. The mechanism of action is through noradrenergic blockade.
 - (2) Antipsychotics may result in QT_c prolongation. Cases of torsades have been reported with use of IV haloperidol. Of the newer antipsychotics, ziprasidone is most associated with QT_c prolongation.
- d. Hyperprolactinemia and its clinical consequences (galactorrhea, amenorrhea) may result from use of antipsychotics, especially risperidone.
- e. Neuroleptic malignant syndrome
 - (1) This uncommon (0.5%–1%) idiosyncratic reaction (usually occurring in the first weeks of treatment or after a dose increase) constitutes a medical emergency. Mortality rates up to 30% can be improved to 10% with early recognition and aggressive supportive treatment.
 - (2) More commonly associated with high-potency neuroleptics, it can also be associated with the low-potency agents or withdrawal of anti-Parkinson agents (eg, discontinuation of L-dopa).
 - (3) Clinical presentation
 - (a) Generalized muscle “lead pipe” rigidity. Occasional patients may also have a superimposed tremor or a cogwheel phenomenon.
 - (b) Mental status change, often presenting as confusion, agitation, or coma
 - (c) Hyperthermia with temperatures often >104°F (40°C), often with diaphoresis
 - (d) Autonomic instability with tachycardia and labile or high blood pressure
 - (4) Laboratory abnormalities
 - (a) Leukocytosis
 - (b) Increased creatine phosphokinase: typically >1,000 IU/L, can be as high as 100,000 IU/L
 - (5) Findings cannot be explained by a medical or neurologic disorder (eg, viral encephalitis), a mental disorder (eg, psychosis), or substance abuse (eg, phencyclidine).
 - (6) Treatment
 - (a) ICU admission is often required.
 - (b) Supportive measures, including IV fluid hydration. Some patients may require mechanical ventilation.
 - (c) The offending agent should be discontinued. Other psychotropic medications (eg, lithium or antidepressants) should also be held.
 - (d) Anticholinergic agents should be avoided; they are not effective and may worsen the reaction by interfering with temperature regulation.
 - (e) Benzodiazepines (eg, lorazepam 1–2 mg) can be used as needed for muscular rigidity or agitation.
 - (f) Dantrolene may reduce rigidity and temperature. Its use is controversial and may cause hepatotoxicity.

5. Antidepressants

- a. Starting antidepressants in the emergency department is not recommended without a high certainty of reliable follow-up. These medications can be dangerous in overdose, and in the short-term may increase suicidal thoughts.
- b. SSRIs (fluoxetine, sertraline, paroxetine, citalopram, escitalopram, fluvoxamine) are the current first-line agents for depressive disorders, premenstrual dysphoric disorder, and anxiety disorders (including panic, obsessive-compulsive, and post-traumatic stress disorders).
 - (1) Adverse effects are generally mild and include nausea, headache, sleep disturbance, and sexual dysfunction.
 - (2) Many of these agents inhibit cytochrome P450, causing drug-drug interactions.
 - (a) Fluoxetine, paroxetine, and venlafaxine (a serotonin norepinephrine-reuptake inhibitor [SNRI]) are P450 2D6 inhibitors and prolong/increase tricyclic antidepressant (TCA) levels, thereby potentiating their cardiotoxic effects. Risperidone, codeine, dextromethorphan, and class IC antidysrhythmics (eg, encainide, flecainide) levels are also increased via this mechanism.
 - (b) Fluoxetine, fluvoxamine, and citalopram are P450 3A4 inhibitors (as is grapefruit juice) and prolong/increase levels of alprazolam, triazolam, clonazepam, and verapamil.
 - (3) Toxicity develops only with large overdoses or when combined with other agents.
- c. SNRIs: inhibit reuptake of both serotonin and norepinephrine, a mechanism of action similar to that of the TCAs without the cardiotoxicity of the TCAs.
 - (1) Venlafaxine: can cause hypertension or orthostatic hypotension
 - (2) Duloxetine: often prescribed for patients with depression or anxiety and a comorbid pain disorder; should not be used in patients with hepatic dysfunction or active alcohol abuse
- d. Other newer antipsychotics
 - (1) Trazodone: may increase serum digoxin and phenytoin levels; orthostasis and rarely priapism can occur.
 - (2) Bupropion: increases norepinephrine and dopamine; causes seizures in overdose and in the predisposed (notably bulimic patients and those with seizure disorders)
- e. TCAs (eg, amitriptyline, nortriptyline, imipramine, and others) were the standard of care through the early 1990s. They have been replaced by the SSRIs because of their many adverse effects:
 - (1) Anticholinergic effects (both peripheral and central)
 - (2) α -adrenergic effects: TCAs act therapeutically by presynaptic norepinephrine reuptake inhibition; sympathetic adverse effects can include both hypertension and orthostatic hypotension with falls (particularly in older patients).
 - (3) Cardiac effects: TCAs are type IA (quinidine-like) antidysrhythmics and delay conduction. In overdose, this causes nonspecific T-wave changes, AV block, \uparrow QT interval, and ventricular tachydysrhythmias, including torsades de pointes and fibrillation.
 - (4) Increased seizure risk
 - (5) Lethality in overdose
- f. Monoamine oxidase inhibitors (MAOIs), eg, phenelzine, tranylcypromine, isocarboxazid, selegiline

- (1) Use of MAOIs has largely been replaced by newer antidepressants; however, emergency physicians should be aware of potential drug-food and drug-drug interactions with these agents.
 - (2) Patients on an MAOI should be on a tyramine-free diet. Consumption of tyramine-containing foods (aged cheeses, cured meats, fava beans, red wine) can lead to a hypertensive crisis.
 - (a) Clinical presentation of impending hypertensive crisis may include severe occipital headache, hypertension, palpitations, chest pain, tachycardia, dilated pupils, diaphoresis, nausea, and vomiting.
 - (b) Treatment
 - i. Phentolamine, an α -antagonist (2.5–5 mg slow IV push, which may be repeated every 10–15 minutes as needed), or sublingual nifedipine
 - ii. β -blockers are contraindicated, because they may worsen hypertension by producing vasoconstriction.
 - (3) Ingestion of an MAOI and other serotonergic agents can lead to life-threatening serotonin syndrome. Patients taking an MAOI should avoid SSRIs, SNRIs, tricyclics, meperidine, dextromethorphan, and MDMA.
 - (4) The antibiotic linezolid is pharmacologically an MAOI. Patients should not be continued on serotonergic agents while taking linezolid.
- 6. Serotonin syndrome**
- a. May occur with drug-drug interactions between SSRIs, MAOIs, and other serotonergic agents (sumatriptan, meperidine, tramadol) or in overdose.
 - b. By Hunter Criteria, serotonin syndrome is diagnosed if the patient took a serotonergic agent and has one of the following:
 - (1) Spontaneous clonus
 - (2) Inducible clonus plus agitation or diaphoresis
 - (3) Ocular clonus plus agitation or diaphoresis
 - (4) Tremor and hyperreflexia
 - (5) Hypertonia and temperature $>100.4^{\circ}\text{F}$ (38°C) plus ocular or inducible clonus
 - c. In addition to the Hunter Criteria, the patient may have additional symptoms including:
 - (1) Altered mental status: confusion, hallucinosis, delirium
 - (2) Autonomic instability: diaphoresis, hyperthermia, orthostasis
 - (3) GI signs: nausea, vomiting, diarrhea
 - (4) Neuromuscular instability: tremor, myoclonus, hyperreflexia
 - (5) Life-threatening seizures and cardiovascular collapse may occur.
 - d. Management
 - (1) Discontinue all serotonergic drugs.
 - (2) Supportive care, including IV fluid hydration and cardiac monitoring
 - (3) Benzodiazepines (eg, lorazepam 1–2 mg) can be used to treat agitation.
 - (4) Cyproheptadine is a serotonin and histamine antagonist that may be used if supportive care does not result in clinical improvement.
 - (5) Patients with severe symptoms may require ICU admission. Patients with moderate symptoms may be admitted with cardiac monitoring until symptoms resolve.

7. Mood stabilizers

a. Mood stabilizers are not usually started in the emergency department given their complications and requirements for close monitoring.

b. Lithium

(1) First used in the late 1800s and a very effective mood stabilizer. It has been shown to reduce risk of suicide in patients with bipolar disorder.

(2) Narrow therapeutic index: therapeutic level is 0.8–1.2 mEq/L; signs of toxicity can be evident at levels as low as 1.5 mEq/L.

(3) Acute toxicity

(a) Can be precipitated by dehydration, diuretics, ACE inhibitors, and NSAIDs

(b) Early signs of lithium toxicity include nausea, vomiting, and diarrhea. Patients then develop sluggishness, confusion, ataxia, and an irregular coarse tremor or myoclonic jerks, followed by seizures and nonconvulsive status epilepticus.

(c) Hemodialysis is indicated for a lithium level >4 mEq/L regardless of clinical status, or for a level >2.5 mEq/L if severe symptoms are present.

(4) Chronic toxicity

(a) Renal toxicity: chronic use of lithium can cause nephrogenic diabetes insipidus, nephrotic syndrome, and chronic interstitial nephritis. Occasionally, this progresses to end-stage renal disease. Creatinine should be monitored in patients taking lithium.

(b) Hypothyroidism also develops as a result of chronic lithium use.

c. Divalproex/valproic acid

(1) Indicated for use in bipolar disorder, seizure disorders, and migraine prophylaxis

(2) Contraindicated in pregnancy given high risk of neural tube defects

(3) Target trough level 50–125 mcg/mL

(4) Valproic acid toxicity

(a) Signs of toxicity are present at levels >180 mcg/mL.

(b) Symptoms include CNS suppression, respiratory depression, hyperammonemia, cerebral edema, tremors, and myoclonus.

(c) May also result in a dose-related hepatotoxicity

(5) Patients on divalproex may develop hyperammonemia and encephalopathy even with normal valproic acid and transaminase levels.

(6) Rare but severe complications include hepatic failure and pancreatitis.

d. Carbamazepine and oxcarbazepine

(1) Indicated for maintenance treatment of bipolar disorder

(2) Oxcarbazepine is used as an alternative to carbamazepine given its better tolerability but has an increased risk of hyponatremia.

e. Lamotrigine

(1) Indicated for treatment of bipolar depression

(2) Risk of Stevens-Johnson syndrome, toxic epidermal necrolysis, and drug reaction with eosinophilia and symptoms (DRESS) syndrome, usually occurring in the first 2–8 weeks of therapy; therefore, slow titration of dose is required.

III. SPECIFIC BEHAVIORAL DISORDERS

A. Psychosis

1. Definition: loss of contact with reality
2. Clinical presentation
 - a. **Delusions:** a "fixed false belief" that is not accepted by the patient's cultural group and that is firmly sustained despite what others believe, and in spite of incontrovertible evidence to the contrary (eg, "The FBI implanted radiotransmitters in my heat ducts").
 - b. **Hallucinations:** a false perception experienced in a sensory modality (usually auditory) occurring while the individual is fully conscious; if they are visual, olfactory, or tactile, suspect a medical- or substance-induced ("organic") disorder.
 - c. **Disorganized speech:** the patient does not make sense, rapidly jumps from one topic to another, or takes long pauses before answering, which is suggestive of thought blocking.
 - d. **Grossly disorganized behavior:** purposeless behavior that does not make sense in context, eg, spreading one's wardrobe across the lawn or masturbating in the emergency room
 - e. **Catatonia:** a neuropsychiatric syndrome characterized by decreased responsiveness to one's environment and either hypo- or hyperactivity
 - (1) A classic presentation includes "waxy flexibility," whereby the examiner can move the patient's limbs and they will stay fixed in the new position.
 - (2) Catatonic excitation resembles phencyclidine (PCP) intoxication and can be extremely dangerous to others, including emergency department staff.
 - f. **Negative symptoms:** flat affect, poverty of speech, loss of volition, interpersonal withdrawal, minor cognitive deficits (acutely psychotic patients are usually oriented)
3. Treatment
 - a. Determination of the underlying cause of an acute psychotic episode may not be possible in the emergency department, but initial treatment and evaluation are the same.
 - b. Establish safety first.
 - (1) Search for weapons (involve security personnel as indicated).
 - (2) For patients who are violent or at imminent risk of becoming violent, manage their acute agitation.
 - c. After behavioral stabilization, proceed immediately to exclude treatable general medical causes of the acute episode (including substance- or medication-induced disorders).
4. Differential diagnosis
 - a. Anatomic: head trauma, stroke, tumor, seizures
 - b. Metabolic: hypoxia, hypoglycemia, ketoacidosis, hyper- or hypocalcemia
 - c. Endocrinologic: hyper- or hypothyroid, hypoparathyroid
 - d. Autoimmune: multiple sclerosis, systemic lupus erythematosus, Hashimoto encephalitis (rare)
 - e. Infectious: HIV, syphilis, encephalitis/meningitis
 - f. Toxic: anticholinergic delirium
 - g. Medications: steroids
 - h. Substances: alcohol intoxication/withdrawal, stimulants, hallucinogens, cocaine

5. Major psychiatric disorders that can present as an acute psychotic episode
 - a. Schizophrenia
 - (1) A chronic disorder affecting ~1% of the population
 - (2) Onset usually in late adolescence/early adulthood
 - (3) Diagnosis requires:
 - (a) At least two of the positive symptoms of psychosis for ≥ 1 month ("positive symptoms" include hallucinations, delusions, disorganized speech, disorganized behavior, or catatonia)
 - (b) Severe impairment in level of functioning
 - (c) Duration of ≥ 6 months of some signs of the disorder
 - (d) Exclusion of substance abuse, medications, or a general medical condition as the cause of the signs and symptoms
 - b. Major depression with psychotic features ("psychotic depression"): a psychosis occurring solely during the course of a depressive episode
 - c. Mania with psychotic features: a psychosis occurring solely during the course of a manic episode
 - d. Schizoaffective disorder: a psychosis that is chronic and often associated with mood symptoms, but it also occurs for significant periods without mood symptoms
 - e. Schizophreniform disorder: a psychosis lasting < 6 months (not during an episode of mood disorder), may be a prodrome to schizophrenia
 - f. Brief psychotic disorder: a psychosis lasting < 1 month (again, not during an episode of mood disorder)
6. Disposition
 - a. Depends on the presence of danger to self or others, the balance between the patient's degree of incapacity, and the social supports available
 - b. Admission is appropriate for:
 - (1) A first psychotic episode
 - (2) Patients who are dangerous to themselves or others (suicidal or assaultive)
 - (3) Grossly debilitated patients
 - (4) Moderately debilitated patients who are without adequate social support in the community
 - c. Some patients with positive symptoms of psychosis may be discharged from the emergency department, particularly if the patient has family or a treatment team that vouches for his or her stability in the community despite active symptoms, and if there are no safety concerns.

B. Depression

1. **A major depressive episode is defined as the presence of depressed mood and/or anhedonia plus at least four of the following symptoms for ≥ 2 weeks**
 - a. Depressed mood (or irritability in children and adolescents)
 - b. Anhedonia (lack of pleasure from daily activities, which may present as withdrawal from usual activities or lack of concern about personal appearance)
 - c. Sleep disturbance: insomnia or hypersomnia
 - d. Fatigue or loss of energy
 - e. Change in appetite (increased or decreased) or unintended weight change

- f. Impaired concentration
 - g. Altered level of activity
 - (1) Agitation or
 - (2) Slowed thoughts, speech, and actions ("psychomotor retardation")
 - h. Sense of worthlessness or guilt
 - i. Suicidal thoughts, plans, threats, actions, or excessive thoughts of death
2. Focal points of the medical history determine whether the patient's mood symptoms are medication-related or due to substance abuse or an underlying medical condition.
 - a. Drugs (prescription, OTC, illicit) known to have depression as an adverse effect
 - (1) Antihypertensives, including methyldopa, β -blockers, reserpine, clonidine
 - (2) Sedative-hypnotics
 - (3) H_2 -blockers (particularly cimetidine)
 - (4) Alcohol
 - (5) Cocaine (a "crash" follows a cocaine binge)
 - (6) Opiates
 - b. Medical causes
 - (1) Neurologic disorders
 - (a) Alzheimer disease/dementia
 - (b) CNS tumor
 - (c) Stroke (especially subcortical)
 - (d) Epilepsy
 - (e) Huntington chorea
 - (f) Multiple sclerosis
 - (g) Parkinson disease
 - (2) Endocrine disorders
 - (a) Addison disease
 - (b) Cushing disease
 - (c) Hypo- or hyperthyroidism
 - (d) Diabetes (and its complications)
 - (3) Other general medical conditions: HIV encephalitis, mononucleosis, renal failure, pancreatic cancer
 3. Focal points of the psychiatric history
 - a. Previous psychiatric illness, including history of psychiatric hospitalizations, suicide attempts, and suicidal ideation
 - b. Recent acute or chronic illnesses (especially life-threatening ones)
 - c. Recent life changes
 - d. Existing social supports or lack thereof
 - e. Always ask about suicidal or homicidal ideation
 4. Treatment and disposition
 - a. The primary goal in evaluating depression is to determine the patient's suicidal potential. An adaptation of the "SAD PERSONS" score correlates well with the need for hospitalization.

Table 36: SAD PERSONS Score

		Points
S	Sex (male)	1
A	Age (<19 or >45 years old)	1
D	Depression (or signs and symptoms of)	2
P	Previous attempts or psychiatric care	1
E	Excessive alcohol or drug use	1
R	Rational thinking (loss of)	2
S	Separated/divorced/widowed/single	1
O	Organized plan or serious attempt	2
N	No social support	1
S	Stated future intent, plan, or mechanism	2

Score: >8 = high risk, 6–8 = intermediate risk, <6 = low risk

- b. Emergent psychiatric consult and possible admission are indicated for patients with a score:
 - (1) >8: almost all patients require admission
 - (2) 6–8: psychiatry consult with follow-up or possible admission
 - (3) <6: may be considered for discharge
- c. Patients appropriate for discharge are often characterized by:
 - (1) A score <6 in a patient who states (and you believe) he or she is not suicidal and who is not psychotic, demented, or intoxicated.
 - (2) A plan for rapid follow-up is established, eg, a referral to an outpatient psychiatrist or counselor.
 - (3) The patient can articulate a plan for safe discharge, eg, by stating that he or she will return to the emergency department if the depression worsens or suicidal ideation occurs or recurs.
 - (4) A supportive environment with family or friends is available, they are informed of the patient's needs, and they are comfortable assisting with the discharge and follow-up.
 - (5) The patient does not have easy access to weapons. Ask if there are firearms in the home; if so, ask the family to remove the weapons, so the depressed person does not have access to them.
- d. Antidepressants require 2–4 weeks to take effect and are not usually started in the emergency department. In some patients, initiation of an antidepressant may increase suicidal thoughts.

C. Suicide

1. Epidemiology

- a. Suicide is the tenth leading cause of death in the United States, claiming more than 38,000 lives in 2010. It is the third leading cause of death among 15- to 24-year-olds and the second leading cause of death among 25- to 34-year-olds.
- b. 0.5% of the general population attempted suicide in 2010, and the ratio of attempts to fatalities is approximately 25:1.

2. Risk assessment for suicide

- a. Anyone who attempts or threatens suicide must be taken seriously and evaluated for suicide risk potential.
 - (1) Ask the patient how he or she feels about having survived an attempt and assess his or her reaction (eg, angry, doesn't care, relieved).
 - (2) Ask directly about the suicidal intent; patients do not find this insulting and it will not "put ideas in their head."
 - (3) Confirm all patient reports with collaterals.
- b. What is the content of the suicidal thoughts? Consider the following in your evaluation:
 - (1) Expressed intent
 - (2) Lethality of the method
 - (3) Likelihood of rescue: was rescue a foregone conclusion, or purely fortuitous?
 - (4) Suicidal thoughts can range from a passive death wish (eg, "I wish I would go to sleep and not wake up, but I would never hurt myself" to having a suicide plan with intent and means to act on this plan. The more detailed the plan, the greater the risk. For a patient who has had a self-injurious act, has anything changed as a result of the act, eg, improved ("I didn't realize people cared") or worsened ("I'm so inadequate, I couldn't even commit suicide successfully").
- c. What are this patient's risk factors for suicide?
 - (1) History of previous suicide attempts conveys the highest risk.
 - (2) Age and ethnicity: white men >65 years old are the demographic group most likely to complete suicide.
 - (3) Gender: men are 3–4 times more likely to complete suicide, whereas women are 3–4 times more likely to attempt suicide. Men are more likely to attempt suicide by a more lethal means (firearms, hanging) than women (ingestion, cutting).
 - (4) Family history of completed suicide
 - (5) Major psychiatric illness, including major depressive disorder, schizophrenia, bipolar disorder, and borderline personality disorder
 - (6) Marital status: those who are separated, divorced, or single are at higher risk
 - (7) Medical illness, particularly chronic illnesses (multiple sclerosis, HIV)
 - (8) Hopelessness: an individual in a personal crisis who sees "no way out" or no recovery from mental illness is at high risk of acting on these thoughts.
 - (9) Recent loss (of job, of relationship, recent death)
 - (10) Alcohol abuse: one-third of completed suicides occur when the individual had consumed alcohol.
 - (11) Additional risk factors include panic attack, agitation, insomnia, and physical pain. These can be addressed while the patient is in the emergency department.
 - (12) In adolescents, the ratio of attempts to completions is 25:1 in girls and 3:1 in boys. Risk is increased with a history of running away, previous suicide threats or attempts, psychiatric disorder, alcohol or substance abuse, and being a lesbian/gay/bisexual/transsexual teen. The presence of a firearm in the household markedly increases (by 5–10 times) the suicide risk.
- d. What factors mitigate suicide risk?
 - (1) Caring for dependent children
 - (2) Strong religious beliefs that deter suicide

- (3) Evidence of planning for the future (being "future-oriented")
- (4) The ability to put in place an action plan for what to do if the suicidal thoughts return
- e. Is there access to lethal means?
 - (1) Firearms are used in 53% of all completed suicides in the United States. Their presence in the home constitutes an independent risk factor for completed suicide. All suicidal patients should be asked about access to firearms.
 - (2) Inquire as to whether the family can safely remove sharp objects and lock up medications.
- f. Consider less obvious presentations.
 - (1) Repeated medical emergencies due to noncompliance with treatment may indicate silent suicide, the act of killing oneself slowly by nonviolent means. This is often unrecognized and is most common in the elderly.
 - (2) "Unintentional" overdoses, wrist lacerations, self-inflicted gunshot wounds, falls from heights, and single-automobile collisions may represent occult suicide, ie, self-destructive acts disguised as accidents.
- 3. Treatment
 - a. Patients at high risk of committing suicide should be hospitalized, and safeguards must be put in place to limit a patient's ability for self-harm while in the hospital. In-hospital suicides are the second most commonly reported sentinel event and occur at a rate of 5–15 per 100,000 admissions.
 - b. After a suicide attempt, medical stabilization of toxicity or trauma is required, followed by the identification and treatment of any comorbid medical condition. Once the patient is medically stabilized, placement in a psychiatric hospital can be considered.
 - c. While in the emergency room:
 - (1) Search for weapons and pills with assistance of security personnel.
 - (2) Constant observation of the patient must be provided, eg, through use of 1:1 sitters.
 - (3) Patients must not be permitted to leave before their assessment has been completed, and if found to be at risk of suicide or to lack decision-making capacity, they must not be permitted to leave against medical advice.
 - (4) Involuntary admission may be required.
 - d. Disposition
 - (1) If the patient's suicide intent is serious, or you are unsure how serious the potential suicide risk is after your evaluation, obtain an emergent psychiatric consultation.
 - (2) While mental health professionals may assist in the evaluation, the emergency physician is required to make an independent judgment and retains final responsibility for medical decisions. In particular, "denial of medical necessity" by a managed care organization or third-party payer does not override the physician's duty to render appropriate and adequate medical care, including, if necessary, involuntary admission.
 - (3) Admission may be required if:
 - (a) The patient will not (or cannot) cooperate with assessment
 - (b) The crisis is ongoing or unresolved
 - (c) After your evaluation (or a psychiatrist's), the patient is still considered a suicide risk
 - (d) When in doubt, err on the side of caution.

- (4) Discharge to outpatient treatment may be considered if:
 - (a) The patient is perceived to be at low risk of acting on suicidal thoughts (eg, if the patient has only passive thoughts of suicide, has no history of attempts, and is able to agree to a "safety plan")
 - (b) The patient has adequate social support, *and*
 - (c) Referral for close outpatient follow up has been arranged.
- (5) Patients who present after an actual suicide gesture (not just thoughts) are almost always referred for inpatient psychiatric treatment. Those with a gesture that is "low lethality" (eg, ingesting a small number of OTC tablets or superficial cuts on the dorsal surface of the arm) and "high rescue" (eg, took the ingestion in front of a family member) might be considered for potential discharge only if the above criteria are already met and the patient's family and outpatient providers are prepared to manage outpatient care.
- (6) Patients deemed high risk may decline voluntary hospitalization and may require an involuntary hold to protect their safety. State laws determine the specific criteria for involuntary hospitalization, the duration of time a patient can be held, and the process for court proceedings for extending the hospitalization.

D. Mania and hypomania

1. A "manic episode" is defined as an excessive, persistently elevated, expansive, or irritable mood and 3 or more of the following symptoms for ≥ 1 week duration:
 - a. Grandiosity or inflated self-esteem
 - b. Decreased need for sleep (without fatigue)
 - c. Pressured speech or increased talkativeness
 - d. "Flight of ideas" (repeatedly jumping from one topic to another loosely related topic in conversation, or a subjective feeling that the mind is "racing")
 - e. Distractibility
 - f. Agitation or increase in goal-directed activity
 - g. Impulsivity ("excessive involvement in pleasurable activities that have a high potential for painful consequences," eg, spending sprees, hypersexual behavior, fights, foolish behavior at work; psychosis is possible, often with paranoid, grandiose, or religious features)
2. A hypomanic episode is of less intense severity, has less impact on the patient's ability to function, and may last < 1 week. Hypomania is not associated with psychosis, and these patients may not require inpatient hospitalization.
3. Focal points of the medical history determine whether the patient's mood symptoms are medication-related or caused by substance abuse or an underlying medical condition.
 - a. Drugs (prescription, OTC, illicit) known to cause manic symptoms
 - (1) Steroids
 - (2) Antidepressants
 - (3) Psychostimulants
 - (4) Phencyclidine (PCP)
 - (5) Caffeine
 - b. Medical causes
 - (1) Hyperthyroidism
 - (2) Cushing syndrome

- (3) Multiple sclerosis
- (4) History of head injury
- (5) Sleep deprivation
- 4. Focal points of the psychiatric history
 - a. Previous psychiatric illness (including outpatient treatment and hospitalizations, especially for previous episodes of depression or mania or for suicide attempts)
 - b. Recent life changes
 - c. Existing social supports or lack thereof
 - d. Homicidal, suicidal, or paranoid ideation
- 5. Treatment and disposition
 - a. Admission will be required if the patient is suicidal, homicidal, or grossly disorganized; if the patient's impulsivity, behavior, or lack of insight poses a danger; or if social resources are inadequate to assure support and follow-up at his or her level of disability.
 - b. A patient may be considered for discharge if:
 - (1) Not suicidal/homicidal, not psychotic, and not overtly disorganized or impaired
 - (2) A psychiatry consult has been obtained, and the patient has close follow-up.
 - (3) The patient agrees to return if the mania worsens or psychosis/suicidality/assaultiveness occurs or recurs.
 - (4) A supportive environment with family or friends is available, they are informed of the patient's needs, and they are comfortable assisting with the discharge and follow up. Patients with mania or hypomania often lack insight into their illness and may not recognize progression of their symptoms.
 - c. Manic patients awaiting psychiatric hospitalization may benefit from initiation of medication.
 - (1) Antipsychotic medications (particularly second-generation antipsychotics such as olanzapine, ziprasidone, and quetiapine) and/or benzodiazepines may be offered proactively to help manage the patient's agitation and restore normal sleep.
 - (2) Mood stabilizers (eg, lithium, carbamazepine, other anticonvulsants) require days to weeks until onset of effect. (For some patients awaiting psychiatric admission, divalproex may be started with a loading dosage of 25–30 mg/kg. Note that divalproex is contraindicated in patients with known/suspected urea cycle disorders or who are pregnant.)

E. Catatonia

- 1. Catatonia is a distinct neuropsychiatric syndrome characterized by motor dysregulation associated with a number of psychiatric, neurologic, and metabolic syndromes. It is often under-recognized in both medical and psychiatric settings.
- 2. Observed features of catatonia range from those that are specific but rare (waxy flexibility or posturing) to those that are common in psychiatric patients (agitation or withdrawal). This range includes:
 - a. Stupor
 - b. Excitement, eg, the patient may have sudden outbursts of talking or dancing
 - c. Mutism, which may appear volitional
 - d. Echolalia (repeating what the examiner says) or echopraxia (copying the examiner's movements)
 - e. Stereotypy: non-goal-directed repetitive movements

- f. Waxy flexibility: the examiner can move the patient's body position and the patient assumes the new position
- g. Negativism: the patient actively resists the examiner's attempts to move the patient
- 3. Both medical and psychiatric etiologies
 - a. Major depression
 - b. Mania
 - c. Chronic psychoses such as schizophrenia
 - d. Stroke
 - e. Cerebral tumors
 - f. Encephalitis
- 4. Malignant catatonia: catatonia accompanied by autonomic instability (hyperthermia, tachycardia, hypertension, or labile blood pressure); can be life threatening
- 5. Treatment
 - a. Benzodiazepines (particularly lorazepam) are the treatment of choice. An initial trial of lorazepam 1–2 mg can be administered orally, IM, or IV. Response should be seen within 1–2 hours. A second dose should be administered if there is no response after 3 hours.
 - b. An initial positive response to lorazepam can confirm the diagnosis in many patients.
 - c. If no response to benzodiazepine treatment, electroconvulsive therapy is the next recommended treatment of choice.
 - d. If the catatonia is associated with an underlying psychiatric disorder, the patient responds to lorazepam, and his or her vital signs remain stable, psychiatric admission may be appropriate.
 - e. Patients with malignant catatonia require medical hospitalization and may require treatment in the ICU.
 - f. Antipsychotics should not be given while the patient is catatonic, because they may increase the likelihood of precipitating neuroleptic malignant syndrome. (Neuroleptic malignant syndrome and catatonia are believed to be related syndromes.)

F. Panic attacks/disorder

- 1. Clinical presentation
 - a. Common: apprehension, tremor, sweats/flushing/chills, shortness of breath, chest tightness or pain, palpitations or tachycardia, difficulty swallowing, nausea, lightheadedness, paresthesias, and a sense of depersonalization, derealization, and feelings of impending doom
 - b. Patients believe that they are suffering from an acute medical problem (and physicians are required to believe that these patients are suffering from an acute medical problem until proven otherwise).
 - c. Attacks usually begin suddenly, sometimes without provocation, and last <30 minutes.
- 2. Diagnostic evaluation
 - a. Vital signs and physical examination
 - b. Exclusion of an underlying medical cause
 - (1) Cardiac (acute myocardial infarction, angina pectoris, mitral valve prolapse)
 - (2) Endocrine (hypoglycemia, hyperthyroidism, hypoparathyroidism, pheochromocytoma)
 - (3) Respiratory (asthma, pulmonary embolism)

- (4) CNS (complex partial seizures, transient ischemic attack, Huntington disease, combined systemic disease/posterolateral sclerosis)
- (5) Medications (methylxanthines, medications with caffeine)
- (6) Substance intoxication (amphetamines, cocaine, hallucinogens, amyl nitrate)
- (7) Substance withdrawal (sedative hypnotics, alcohol)
- c. A history of previous medical and psychiatric illnesses, excessive caffeine use, prior attacks, and any possible precipitating stressful event
- 3. Treatment
 - a. Referral for behavioral health treatment, which may include:
 - (1) Specialized cognitive-behavioral therapy
 - (2) Short-term management with high-potency benzodiazepines (lorazepam, alprazolam) while awaiting antidepressant efficacy
 - (3) Long-term management with antidepressants
 - (4) β -blockers, buspirone, and antihistamines are ineffective.
 - b. Advise patients to avoid alcohol and caffeine and to get adequate sleep and exercise.
 - c. Immediate interventions aimed at reducing hyperventilation (such as having the patient lock his hands behind his neck) improve hypocapnia and respiratory alkalosis, and thus help to resolve paresthesias, lightheadedness, and derealization/depersonalization, as well as provide a reassuring sense of self-control.
 - d. The most useful intervention is the communication to the patient that any potentially life-threatening medical disorder has indeed been excluded. During a panic attack, the patient feels that he or she is about to die (and, indeed, symptoms can be indistinguishable from an acute MI). This is itself frightening, and the emotional reaction to the perceived life threat causes further sympathetic arousal, thus further exacerbating symptoms.

G. Somatoform disorders

- 1. Somatoform disorders include conversion disorder, somatization disorder, hypochondriasis, and pain disorder. These disorders are all characterized by psychological conflict that manifests either as physical symptoms or as concern about symptoms or illness.
 - a. The link between psychological distress and physical symptoms may be apparent to the physician but is not realized by the patient.
 - b. This is not malingering. Malingering is an intentional faking of symptoms for conscious personal gain such as obtaining drugs, avoiding legal responsibility, or gaining shelter in the hospital.
 - c. This is not a factitious disorder in which symptoms are intentionally produced or feigned but with no motivation or incentive other than to assume the "sick role" (primary gain).
 - d. Psychiatric consults may be of limited use for somatoform patients. The patient will not want (and may resent) this suggestion, because the somatoform symptom serves the purpose of masking a psychological conflict.
- 2. **Conversion disorder**
 - a. **A psychological conflict "converts" into an acute loss of neurologic function that allows the patient to avoid or resolve the conflict; typical examples include loss of voluntary motor or sensory function (eg, paralysis of one or both legs), blindness, or nonepileptic seizures ("pseudoseizures").**
 - b. **Diagnostic criteria**

- (1) Symptom(s) or deficit(s) of voluntary sensory or motor function suggesting a neurologic (or other medical) condition, eg, stroke
- (2) These symptoms/deficits are judged to be due to psychological factors, because conflicts or stressors precede their onset/exacerbation.
- (3) They are not intentionally produced or "faked."
- (4) No demonstrable source from a general medical disorder, substance use/abuse, or culture-specific pattern (This is a diagnosis of exclusion and should not be made without an appropriate medical evaluation to exclude physiologic causes of the symptoms.)

c. Clinical presentation

- (1) The patient appears healthy and may be less concerned about his or her symptoms than you would expect ("la belle indifférence").
- (2) The physical symptoms typically developed suddenly and involve a voluntary muscle function (most common) or loss of sensation. They also tend to have a relationship to the conflict they "solve" (eg, a man who hates his manual labor job develops "paralyzed" legs at home Sunday night). Ask the patient what it is that he or she cannot do now and what it was that he or she was doing when the symptoms began.

d. Treatment

- (1) Perform a detailed history and physical examination, and obtain diagnostic studies as indicated to exclude medical/surgical pathology.
- (2) Once the evaluation is completed, assure the patient that a serious medical problem has not been discovered and suggest that the symptom will resolve. Most cases of conversion disorder resolve without intervention.
- (3) About 25%–50% of patients initially diagnosed with conversion disorder are eventually found to be suffering from significant medical pathology (systemic lupus erythematosus, multiple sclerosis, polymyositis, thyroid disease, porphyria, hypo- or hyperparathyroidism); therefore, referral for thorough evaluation and ongoing follow up is a must.

3. Somatization disorder

- a. Patients present repeatedly to medical practice settings, including the emergency department, with multiple physiologic complaints. Psychologic conflict is manifest as physical complaints that gain the focus of the patient's attention. The review of physical systems will have positive findings in multiple organ systems.
- b. To meet diagnostic criteria, patients must have pain complaints on four body sites, two GI symptoms, one pseudoneurologic symptom, and one sexual symptom.
- c. Treatment is coordinated through an involved primary care physician. Appointments are scheduled at regular intervals, and discussions restricted to a limited number of complaints.
- d. Medical evaluation of physical symptoms should be thoughtfully selected and guided by objective findings.

4. Hypochondriasis

- a. The patient intensely believes that he or she has a specific disease such as cancer or HIV. The patient presents with "evidence" of the disease and repeatedly requests testing.
- b. In hypochondriasis, the patient is concerned with disease, while in somatization disorder, the patient is concerned with symptoms.

5. Somatoform pain disorder

- a. A patient presents with one or more pain symptoms that cause clinically significant distress or impairment.
 - b. Psychological factors are believed to have an important role in the onset, severity, exacerbation, or maintenance of the pain. The patient may also have an underlying physiologic reason for the pain, but the pain is usually disproportionately more than what would be expected.
 - c. The patient is experiencing real pain (ie, it is not faked) and usually believes it is due to a physical problem.
 - d. This is difficult to detect in the emergency department. The physician may be concerned that the patient is "drug seeking" because of the disproportionate level of pain.
6. Psychophysiologic reactions
- a. A physical condition brought on by an event that is associated with a heavy emotional overlay, eg, nausea and diarrhea that occur before a performance. This is not a somatoform disorder but another example of a link between psychological factors and physical symptoms.
 - b. Symptoms are real although engendered unconsciously; physiologic illness develops to express or resolve emotional conflict without the patient realizing it. These reactions can also be understood as "overflow" autonomic activation in response to a stimulus.
 - c. Treat the medical condition without confronting the patient with the psychogenic cause.
 - d. The reactive condition is not produced voluntarily or intentionally and is, in fact, realistically distressing/handicapping to the patient. Gently suggest a stress-related emotional-physical connection. It may be possible to illustrate this by having the patient reenact the situation.
 - e. These patients may benefit from psychotherapy or biofeedback.
7. Techniques that distinguish neurologic from psychogenic deficits
- a. Bowles and Currier test (sensation): Have the patient extend his or her arms, crossing them at the wrists, so the palms are facing each other; then have him or her interlock the fingers and rotate the hands inward toward the chest. In this position, false responses to sensory testing are difficult.
 - b. Hoover test (motor): Cup your hands under the heels of the patient, and ask him to raise his or her normal leg. With pseudoparalysis, the other leg will push downward.
 - c. Gray test (pain): Patients with abdominal pain that is psychological in origin will close their eyes on palpation of the abdomen (most patients with organic pain watch your hand in anticipation of pain).
 - d. Bell phenomenon (coma): In true coma, the eyes remain neutral when the lids are opened; in conversion, the eyes are diverted upward.
 - e. Corneal reflex: intact with pseudoseizure
 - f. Optokinetic drum: nystagmus in pseudoblindness

H. Insomnia and sleep disorders

- 1. Insomnia is one of the most common medical complaints. Patients report difficulty falling or staying asleep, or not feeling rested the following day.
- 2. Myriad causes of sleep disturbance exist and include the following:
 - a. Psychophysiologic insomnia (also called primary insomnia): the patient becomes increasingly concerned about the delay in falling sleep, which leads to an increase in anxiety and greater delay in sleep onset.
 - b. Extrinsic factors

- (1) Poor sleep hygiene (eg, TV and lights remain on at night)
- (2) Shift-work sleep disorder
- (3) Jet lag
- c. Psychological contributors include mood disturbances (eg, mania or major depression) and anticipatory anxiety (eg, about an exam).
- d. Alcohol and other substances directly disrupt the sleep-wake cycle.
- e. Medical problems can impair sleep, eg, obstructive sleep apnea, restless legs syndrome, orthopnea, physical pain.
- 3. Treatment
 - a. Treatment of insomnia focuses on identification and treatment of the underlying pathology (eg, obstructive sleep apnea, depression). However, this is not always possible in the emergency room setting.
 - b. Sleep hygiene education should be provided. Patients should avoid caffeine, exercise, and alcohol in the evening, and they should go to bed and rise at the same time every day. The bedroom should be restricted to use for sleep and sex, and televisions should be removed from the room.
 - c. Sleep aids can be useful for acute insomnia. They generally should not be used for >2 weeks, because tolerance and withdrawal can develop.
 - (1) Zolpidem 5–10 mg is often used to treat insomnia. Adverse effects include increased risk of parasomnias (eg, sleep walking).
 - (2) Long-acting benzodiazepines are used for those with middle-of-the-night insomnia.
 - (3) Melatonin, a hormone produced by the pineal gland that triggers natural onset of sleep, is often recommended for jet lag. It is available OTC.
 - (4) OTC sleep aids (eg, diphenhydramine) have limited effectiveness and make use of sedating, antihistamine effects.
 - d. Patients presenting to the emergency department with insomnia should follow up with their primary care provider.

I. Eating disorders

- 1. Anorexia nervosa
 - a. Prevalence is approximately 1% in Western societies; women are most commonly affected (95%), particularly those from middle and upper socioeconomic classes.
 - b. Onset generally occurs between 12 and 18 years of age and usually follows an episode of severe dieting or other weight loss.
 - c. The central characteristic is a preoccupying fear of being overweight that does not diminish with weight loss. It may or may not be associated with purging behaviors (self-induced vomiting, or laxative or diuretic abuse).
 - d. Prognosis improves with earlier onset, diagnosis, and treatment. Chronicity increases morbidity.
 - e. Mortality at 10-year follow-up is 7%; at 30-year follow-up, 18%.
 - f. Diagnostic criteria
 - (1) Weight <85% of ideal body weight.
 - (2) Intense fear of gaining weight or becoming "fat" despite being underweight
 - (3) Distorted body image, overconcern with weight/ shape, or denial of seriousness of current cachexia
 - (4) Amenorrhea for ≥ 3 consecutive menstrual cycles

- g. Patients with anorexia nervosa are unmotivated for treatment; they notoriously resist detection (frank deception is common, eg, as in surreptitiously carrying weights in underwear). Clues include:

- (1) Unexplained growth retardation
- (2) Unexplained weight loss
- (3) Unexplained primary or secondary amenorrhea
- (4) Hypercholesterolemia and/or carotenemia while underweight
- (5) Exercise abuse
- (6) Vulnerable vocation/activity (eg, dancers, gymnasts, models, wrestlers, jockeys)

- h. Treatment

- (1) Correction of metabolic derangements; hypokalemia and hyperkalemia can be especially dangerous, because arrhythmia may be induced.
- (2) Outpatient treatment may be attempted if there is:
 - (a) Restriction and weight loss <3 months duration
 - (b) Good family support system
 - (c) Patient compliance
- (3) Medical admission is indicated when:
 - (a) Weight loss >25% of ideal body weight (if the weight loss has been rapid or has occurred in a still-growing adolescent, in which case, a weight loss >15% ideal body weight is sufficient to hospitalize)
 - (b) Hypokalemia
 - (c) Hyponatremia
 - (d) Bradycardia with heart rate <45 beats per minute
- (4) Admission (either medical or psychiatric) may be indicated when:
 - (a) Severe psychiatric illness or comorbid suicidality
 - (b) Worsening ability to control self-induced vomiting, binge eating, or diuretic/laxative use, which may be life threatening
 - (c) The patient's food refusal or obsessive thoughts leads him or her to be uncooperative with lower levels of treatment

- i. Risk of refeeding syndrome

- (1) Refeeding syndrome is a potentially fatal shift in fluids and electrolytes when nutrition is reinitiated in a malnourished patient.
- (2) Electrolyte abnormalities include hypophosphatemia, hypomagnesemia, and hypokalemia. This can result in volume overload and may precipitate heart failure. A normal heart rate in a patient who has been bradycardic may be a harbinger of the refeeding syndrome.
- (3) Patients who have experienced rapid weight loss are at risk of refeeding syndrome within the first few days of starting nutrition. The risk is higher with parenteral nutrition but may also occur with enteral nutrition.
- (4) To prevent refeeding syndrome, a nutritional consult should be obtained and nutrition should be reinitiated slowly. Serum electrolytes should be checked daily.
- (5) Thiamine should also be supplemented to reduce risk of Wernicke encephalopathy.

2. Bulimia nervosa

- a. Prevalence is ~5% in young adult women in Western societies.

- b. Onset is usually at 17–25 years of age and follows an episode of anorexia in 30% of patients.
- c. Binge episodes are defined as eating an unusually large amount of food accompanied by a sense of loss of control. The patient may eat several thousands of calories at once. This is often concealed from family and physicians.
- d. Purging is defined as “inappropriate compensatory behavior in order to prevent weight gain,” including self-induced vomiting, fasting, abuse of laxatives/diuretics/enemas, and/or excessive exercise.
- e. Weight may fluctuate but is often in the normal range.
- f. Diagnostic criteria
 - (1) Binge-eating and purging must both occur, on average, twice a week over a 3-month period. (Less severe patterns also deserve medical and psychiatric attention.)
 - (2) Self-evaluation is overly dependent on weight and body shape.
 - (3) Symptoms do not occur exclusively during an episode of anorexia nervosa.
- g. Medical complications
 - (1) Episodes of starvation can produce all of the metabolic complications of anorexia nervosa.
 - (2) Self-induced vomiting can cause:
 - (a) Dental erosion and gingivitis
 - (b) Parotid and submandibular gland enlargement
 - (c) Knuckle callouses, oral lacerations and contusions
 - (d) Dysphagia, hematemesis
 - (e) Esophageal rupture, subcutaneous emphysema, or pneumomediastinum (all rare)
 - (f) Severe hypokalemia and hypovolemia
 - (g) Electrolyte abnormalities can cause cardiac dysrhythmias and sudden death.
 - (h) Hyperamylasemia
 - (3) Ipecac abuse can cause cardiomyopathy and dermatomyositis.
 - (4) Laxative abuse can cause:
 - (a) Dehydration
 - (b) Hypokalemia
 - (c) Constipation, diarrhea, abdominal cramping, and bloating
 - (d) Cathartic colon: loss of colonic physiologic peristalsis, such that large doses of laxatives become required to effect fecal movement. This condition is not entirely reversible and may require colectomy.
 - (5) Diuretic abuse can cause:
 - (a) Dehydration
 - (b) Hypokalemia, hypercalcemia, hyperuricemia, hypomagnesemia, and hyponatremia
 - (6) Binge eating after a period of starvation has further complications.
 - (a) Acute gastric distention (even rupture)
 - (b) Acute pancreatitis (mortality 10%)

h. Treatment

- (1) Normotensive, hypokalemic, hypochloremic metabolic alkalosis is a typical manifestation of chronic purging. The patient has usually adapted physiologically to this state. Overzealous efforts at treatment frequently lead to overcorrection and fluid overload (which can be dangerous).
- (2) Nutritional rehabilitation: normalization of the eating pattern is the primary consideration.
- (3) Medical admission is indicated for:
 - (a) Vital sign abnormalities
 - (b) Hypokalemia
 - (c) Hyponatremia
 - (d) Comorbid medical disorder (eg, diabetes, particularly in a patient who is withholding insulin as a means of losing weight)
- (4) Psychiatric admission may be indicated when:
 - (a) Severe psychiatric illness or comorbid suicidality
 - (b) Worsening ability to control self-induced vomiting, binge eating, or diuretic/laxative use, which may be life threatening
 - (c) The patient's bingeing, purging, or obsessive thoughts leads him or her to be uncooperative with lower levels of treatment.
- (5) Definitive treatment after medical stabilization requires cognitive-behavioral psychotherapy and/or antidepressant medication (an SSRI); combining both psychotherapy and medication may enhance treatment efficacy.

J. Intoxication and withdrawal

1. Intoxication: impairment of judgment, perception, attention, emotional control, and psychomotor activity produced by recent ingestion of a drug
 - a. Clinical presentation is predominately impaired judgment and motor coordination. With increasing severity, delirium, stupor, coma, and death may occur.
 - b. Intoxication should be included in the differential diagnosis of any patient with an altered state of consciousness.
2. Urine drug screens do not have perfect sensitivity. Watch for false-negatives (oxycodone and methylphenidate are often not detected) or false-positives (eg, pseudoephedrine showing positive for amphetamines).
3. Treatment should be guided by the following principles:
 - a. Provision of general supportive measures that include the ABC priorities
 - b. Specific measures to remove toxic substances or substances that have been ingested in toxic amounts
 - c. Exclusion of other serious medical, surgical, or psychiatric disorders
 - d. Appropriate referral—substance abuse is a chronic and episodic disorder, and effective treatment is available.
4. Alcohol or other sedative-hypnotics (benzodiazepines)
 - a. Intoxication
 - (1) Symptoms include an initial feeling of euphoria and disinhibition, followed by slurred speech and ataxia. New memories are not encoded into memory (anterograde amnesia). More severe intoxication may present with either agitation or sedation.

- (2) Degree of impairment depends on total alcohol consumption and also previous exposure (tolerance). "Legal limit" for driving in most states is a blood alcohol level of 0.08 g/dL.
- (3) If tranquilization is required, use of additional sedativehypnotic medications (such as lorazepam) should be avoided. An antipsychotic (eg, haloperidol 5 mg or chlorpromazine 25–50 mg orally or IM every 30–60 minutes) is a safer choice because these contribute little to respiratory depression.
- (4) Treatment of alcohol intoxication requires placement in a secure setting and allowing time to pass for the patient to metabolize the alcohol. If large quantities of alcohol have been rapidly ingested, intubation may be required for airway protection.

b. Withdrawal

- (1) For a person with an average rate of alcohol metabolism, the blood alcohol level will drop by ~0.02 g/dL per hour.
- (2) A seasoned alcoholic may begin to show alcohol withdrawal with a blood alcohol content well above the "legal limit."
- (3) Withdrawal symptoms: 6–24 hours from the last drink, patients experience a high-amplitude tremor and autonomic hyperactivity (nausea, tremor, hypertension, tachycardia, hyperreflexia, sleep disturbances, and anxiety)
- (4) Withdrawal seizures: generalized tonic-clonic seizures that usually occur 12–48 hours from last drink. Indications for CT of the head in patients who have seizures:
 - (a) New-onset seizure
 - (b) Focal seizure
 - (c) Focal neurologic deficit
 - (d) Status epilepticus
 - (e) With significant head trauma
- (5) Alcohol withdrawal delirium (delirium tremens): delirium (characterized by an altered level of consciousness, disorientation, confusion, and often visual hallucinations) with onset 3–5 days after last drink. Autonomic instability with gross tremor, fever, incontinence, mydriasis, and frightening visual hallucinations may occur, leading to substantial mortality and often requiring ICU admission.
- (6) Alcohol withdrawal is treated with benzodiazepines. Lorazepam does not require metabolism by hepatic oxidation and is preferred for patients with hepatic dysfunction. Longer-acting agents (chlordiazepoxide or diazepam) are also used. Benzodiazepine dosages are based on the patient's Clinical Institute Withdrawal Assessment (CIWA) score.
 - (a) These patients are generally volume-depleted, hypomagnesemic, malnourished, and hypoglycemic. Provide IV hydration (D5NS), thiamine 100 mg, and magnesium 2–4 g.
 - (b) Do not administer glucose or other carbohydrate before replacing thiamine (IV); doing so can precipitate Wernicke encephalopathy and/or Korsakoff syndrome with brain damage that is frequently permanently disabling.
- (7) Disposition is determined by the patient's stage of withdrawal and history of complicated withdrawals.

- (a) Patients in early withdrawal (autonomic hyperactivity) who respond to emergency department treatment can often be managed as outpatients or in a dedicated detoxification/substance abuse center. These patients may require 1–2 days of therapy with a long-acting benzodiazepine. This is controversial, because these medications may mask symptoms of a medical problem or may be sold on the street. Close follow-up is required.
 - (b) Patients may require medical admission for alcohol withdrawal if they have a history of complicated withdrawals (including delirium or seizures) or if their presenting symptoms cannot be controlled with oral benzodiazepines. The presence of seizures, hallucinations, delirium, or signs of Wernicke encephalopathy merits a medical admission.
- 5. Opioids (including heroin, oxycodone, hydrocodone, hydromorphone, morphine, meperidine, opium, methadone)
 - a. Intoxication
 - (1) Toxidrome includes somnolence, decreased respiration rate, decreased tidal volume, decreased bowel sounds, and miotic pupils. The absence of miotic pupils does not exclude opioids as the cause of a change in mental status.
 - (2) Usual presentation in the emergency department is of a person who has overdosed and may have received naloxone in the field. Accidental overdose of opioids is rising rapidly as a cause of death in the United States.
 - (3) Naloxone is a competitive opioid antagonist that quickly reverses the effects of opioid intoxication. It can be given IV, IM, SC, or by nasal spray. Usual doses are 0.4–2 mg, and it can be repeated up to 10 mg. Effects of naloxone last for 30–60 minutes, so repeated dosing may be needed.
 - (4) Opioid-addicted individuals may also present to the emergency department with medical complications of their drug use, eg, cellulitis or abscesses from “skin popping” (injecting heroin subcutaneously).
 - b. Withdrawal
 - (1) Unlike alcohol withdrawal, opioid withdrawal is unlikely to be life threatening.
 - (2) Symptoms include muscle aches, abdominal cramps, malaise, nausea, yawning, rhinorrhea, piloerection, diarrhea, sweats, and chills.
 - (3) Vital signs are often normal, although the patient may be tachycardic or hypertensive due to physical discomfort or pain.
 - (4) Opioid withdrawal is treated symptomatically, for example:
 - (a) Clonidine to decrease adrenergic tone, which results in many withdrawal symptoms
 - (b) Ibuprofen for muscle aches
 - (c) Dicyclomine for abdominal cramps
 - (d) Imodium for diarrhea
 - (e) Buprenorphine (sometimes)—Caution should be used in giving buprenorphine too soon after last opioid use, in particular with long-acting opioids such as methadone. Because it is a partial agonist that binds strongly to the mu receptor, buprenorphine may precipitate withdrawal if given too soon.
 - (5) Medical admission is usually not warranted.
- 6. Cocaine, bath salts, and other psychostimulants (amphetamine, methamphetamine, methylphenidate)
 - a. Intoxication

- (1) Cocaine can be snorted as powder, injected, or smoked as crack-cocaine. Effects usually last <30 minutes depending on method of use.
- (2) Amphetamines may be taken as pills, crushed, snorted, or injected.
- (3) Bath salts are synthetic cathinone derivatives that are increasingly used as drugs of abuse. Commonly used drug screens do not currently screen for bath salts.
- (4) Use of these drugs leads to a sympathomimetic toxidrome with hypertension, tachycardia, mydriasis, hyperalertness, and diaphoresis. Symptoms include a feeling of euphoria, increased alertness, and increased energy. Anxiety, paranoia, and restlessness are also common.
- (5) Frequent users of cocaine or psychostimulants may develop chronic hallucinations that are sometimes mistaken for a primary psychotic disorder.
- (6) Cocaine is an etiologic factor in many chest pain presentations and may lead to myocardial infarctions even in young patients.
- (7) Sensorium is usually clear, although delirium is possible.
- (8) Seizures may occur.
- (9) Benzodiazepines are recommended to treat the agitation associated with use of these drugs.
- (10) Medical admission may be necessary for medical sequelae (eg, sustained tachycardia, chest pain, or seizures).

b. Withdrawal

- (1) Physiologic dependence on cocaine and psychostimulants does not occur, but individuals can become psychologically dependent on the drug.
- (2) Cessation of drug use results in dysphoria, hypersomnolence, hyperphagia, irritability, increased dreaming, and drug craving. Patients coming off a drug binge may "crash" and experience a period of depression and suicidal ideation.
- (3) Inpatient detoxification centers that may treat alcohol, benzodiazepine, or opioid withdrawal usually do not treat patients seeking help for addiction for cocaine or other psychostimulants.

7. Phencyclidine (PCP) intoxication

- a. Symptoms may include muscle tension, tachycardia, hypertension, hyperthermia, drooling, nystagmus (horizontal, vertical, and rotary), analgesia, and loss of proprioception with ataxia. Severe hyperthermia, rhabdomyolysis, seizures, and coma may be seen with large ingestions.
- b. Bizarre behavior and extreme violence are possible; emergency department personnel have been fatally battered by such patients.
- c. High-potency benzodiazepines are the initial agents for agitation and seizures (eg, lorazepam 1–2 mg orally or IM every 30–60 minutes).

K. Delirium and dementia

1. Delirium is an acute problem, characterized by an alteration in mental state and consciousness, that is due to an underlying medical cause. In contrast, dementia is a set of chronic, progressive neurodegenerative disorders, including Alzheimer disease, vascular dementia, and Lewy body dementia.
2. Patients with dementia are predisposed to bouts of delirium. For these patients, differentiating between delirium and dementia requires knowledge of the patient's baseline mental state.

3. Delirium
 - a. Diagnostic features
 - (1) Disturbance of consciousness with reduced ability to focus, sustain, or shift attention.
 - (2) Cognitive impairment (eg, memory deficit, disorientation, dysphasia) or perceptual disturbance
 - (3) Acute onset (hours to days) with diurnal fluctuation of symptoms
 - (4) Physiologic result of a general medical condition
 - b. Clinical presentation
 - (1) Fluctuating level of consciousness and alertness
 - (2) Trouble paying attention; ask the patient to state the days of the week backward (a delirious patient will often state them forward)
 - (3) May be characterized by hyperactive or hypoactive motor activity
 - (4) Visual hallucinations (common)
 - c. Search for underlying cause.
 - (1) Metabolic disorders
 - (2) CNS injury or mass lesion
 - (3) Infection (CNS or elsewhere)
 - (4) Medication effects/interactions
 - (5) Drugs, heavy metals, toxins, poisons
 - d. Treatment revolves around correction of underlying cause.
 - (1) Patients with delirium are hospitalized in a medical facility because of their underlying medical illness.
 - (2) If the cause of delirium is not determined in the emergency department, these patients are hospitalized to determine the underlying cause.
4. Dementia: a global and progressive impairment of cognitive function without an alteration of consciousness
 - a. Diagnostic features: multiple cognitive deficits with both
 - (1) Memory impairment (either ability to learn or recall) and
 - (2) At least one other cognitive disturbance
 - (a) Aphasia (language deficits)
 - (b) Apraxia (inability to perform known tasks)
 - (c) Agnosia ("not knowing")
 - (d) Impaired executive function (planning, organizing, sequencing, abstracting)
 - b. Clinical presentation
 - (1) Gradual onset (months or years)
 - (2) Disturbance of recent memory may be the first sign.
 - (3) Dementia may become acutely worse in response to another illness, change in living situation, or medications.
 - (4) Common causes of reversible dementia
 - (a) Metabolic and endocrine disorders
 - i. Hypothyroidism
 - ii. B₁₂ deficiency
 - (b) Normal pressure hydrocephalus

- (c) Depression (depressive "pseudodementia") should be suspected if onset was relatively acute and accompanied by vegetative signs of depression (eg, loss of appetite and weight, sleep disturbances, etc). Reversible medical/surgical causes must be excluded.
 - (d) Medication effects
 - i. Anticholinergic medications
 - ii. Polypharmacy
 - iii. Incorrect medication usage (demented patients may take medications incorrectly)
- c. Treatment
 - (1) Initial treatment focuses on correcting reversible causes of dementia and reviewing the drug regimen to identify medications that may exacerbate the cognitive symptoms.
 - (2) The progression of the dementia may be slowed by modulating risk factors (eg, treating hypertension in vascular dementia) or use of anticholinesterase inhibitors (eg, donepezil).
 - (3) Discussions about advance directives, power of attorney, code status, and long-term care wishes should be started (if not done already) at the onset of the diagnosis, while patients still have capacity to make these decision for themselves.
- L. "Medical mimickers" of psychiatric illness (medical illnesses that manifest psychiatric symptoms)
 - 1. Acute intermittent porphyria
 - a. Presents as episodic psychosis (with agitation and hallucinations) and acute abdominal pain
 - b. Neurologic symptoms are due to accumulation of early heme precursors due to a deficiency of porphobilinogen deaminase.
 - c. Attacks are precipitated by drugs, hormones, and diet that stress the hepatic aminolevulinic acid synthase and cytochrome P450 systems. Offenders include barbiturates and other anticonvulsants, sulfonamide antibiotics, low-calorie and low-carbohydrate diets, alcohol abuse, progesterone/progestins, and nonspecific severe stress.
 - d. Diagnosis is made by laboratory quantification of urinary aminolevulinic acid and porphobilinogen, which are markedly increased during attacks and between recurring attacks. Urine dipstick urobilinogen tests are nonspecific, and urine exposed to sunlight turns dark or red in color.
 - e. Treatment is primarily supportive. Patients may be admitted and observed for neurologic complications.
 - 2. Hyperthyroidism
 - a. Symptoms include anxiety, emotional lability, weakness, tremor, palpitations, heat intolerance, increased perspiration, and weight loss despite a normal or increased appetite.
 - b. Physical examination may be notable for hyperactivity, rapid speech, and lid retraction, which may make the patient appear to be staring.
 - c. Diagnosis is made on the basis of thyroid function tests. Thyroid-stimulating hormone is usually <0.05 mIU/L, and free T_4 and T_3 are increased.

3. Hypothyroidism

- a. Symptoms include fatigue, slow movement, and slow speech, which can be mistaken for symptoms of depression. Additional symptoms include cold intolerance, constipation, weight gain, delayed relaxation of deep tendon reflexes, periorbital edema, or exophthalmos.
- b. Hashimoto encephalopathy is a syndrome characterized by confusion and an altered mental state that is associated with Hashimoto thyroiditis. Up to one-third of patients also have psychotic symptoms. Laboratory studies are notable for increased antithyroid peroxidase antibody (TPOAb) and/or antithyroglobulin antibody (TgAb).

4. HIV/AIDS

- a. HIV-associated dementia, also known as AIDS dementia complex, occurs usually in patients with CD4 <200, older age, and medical comorbidities.
- b. Minor cognitive motor disorder has less severe cognitive dysfunction than HIV-associated dementia, occurs earlier in the illness course, and may be mistaken for a psychiatric illness.
- c. Patients with HIV also have greater susceptibility to delirium, have high rates of comorbid psychiatric illness, and can develop CNS lesions such as toxoplasmosis and lymphoma.

5. Wilson disease (hepatolenticular degeneration)

- a. Disorder of cellular copper transport, leading to accumulation of copper in organs (notably liver, brain, cornea)
- b. Patients present with liver disease, neurologic symptoms, or psychiatric symptoms. They may also have dysarthria, gait abnormalities, tremor, or other abnormal movements.
- c. Psychiatric symptoms are manifestations of frontal lobe impairment and may include impulsivity, mood lability, personality change, depression, or psychosis. Other patients may develop a dementia-like syndrome.
- d. Diagnosis is by low ceruloplasmin level, Kayser-Fleischer rings, and increased 24-hour urinary copper excretion.

PSYCHOBEHAVIORAL DISORDERS: PRACTICE CLINICAL SCENARIOS

Answers immediately follow the practice clinical scenarios.

Scenario A

Presentation: A middle aged patient had his last alcoholic drink 3–5 days before presentation. He has tremors, confusion, visual or tactile hallucinations, agitation, and autonomic hyperactivity including tachycardia, diaphoresis, anxiety, hypertension, and insomnia.

Physical examination: The patient has signs of liver disease, malnutrition, and a history of trauma.

What is the diagnosis?

Scenario B

Presentation: A patient presents with sad mood, loss of interest in usual activities, decreased appetite, weight loss, insomnia, and hopelessness. There may or may not have been a precipitating event that led to these symptoms.

What is the diagnosis?

Scenario C

Presentation: A patient develops confusion, stiffness, and fever shortly after starting a new antipsychotic medication.

Physical examination: The patient has “lead-pipe” rigidity. Altered mental status may range from coma to agitation. Body temperature is $>104^{\circ}\text{F}$ (40°C), and there is tachycardia and hypertension or labile blood pressure.

What is the diagnosis?

Scenario D

Presentation: A young patient presents believing he is having a heart attack. Symptoms include tachycardia, palpitations, chest pain, shortness of breath, diaphoresis, and a sense of doom. Symptoms escalate over ~10 minutes, and the full episode lasts <30 minutes.

What is the diagnosis?

Scenario E

Presentation: A patient is brought to the emergency department by police because of bizarre behaviors, agitation, and hyperactivity. The patient has not slept recently and talks very loudly, quickly jumping from one topic to the next. The thought content may be notable for grandiose, sexual, or religious themes.

What is the diagnosis?

ANSWERS TO PRACTICE CLINICAL SCENARIOS

Scenario A

Diagnosis: delirium tremens (alcohol withdrawal delirium)

Diagnostic evaluation: Laboratory findings may suggest chronic alcohol use. AST and ALT are increased, often in 2:1 ratio. The MCV is >100 fL, and there is thrombocytopenia.

Management: The patient may require ICU admission and mechanical ventilation depending on severity. Benzodiazepines are the mainstay of treatment; lorazepam IV should be scheduled as a standing order, with additional lorazepam given to target symptom severity. Untreated, mortality is ~20%.

Scenario B

Diagnosis: major depression

Management: Evaluation in the emergency department must focus on assessing for risk of suicide, homicide, psychosis, and disability. Gathering information from family members is an essential part of this assessment.

Treatment decisions in the emergency room focus on disposition and referral. Antidepressants are not usually started in the emergency department unless close follow-up can be assured. Antidepressants may lead to an increase in suicidal thoughts in some patients. The patient will require psychiatric hospitalization if he or she is unable to maintain safety of self and others, or is grossly impaired.

Scenario C

Diagnosis: neuroleptic malignant syndrome

Diagnostic evaluation: The WBC count and creatine phosphokinase level are increased.

Management: Discontinue all offending agents and give hydration IV. ICU admission may be required. Avoid anticholinergic medications. Use benzodiazepines for agitation. High-potency neuroleptics such as haloperidol and fluphenazine are most likely to lead to neuroleptic malignant syndrome.

Scenario D

Diagnosis: panic attack

Diagnostic evaluation: The focus should be on excluding medical causes of this presentation, particularly for a patient who has never previously had a panic attack. No other cause for the symptoms is found, and the symptoms resolve.

Management: Benzodiazepines may relieve the symptoms of a panic attack. A patient with recurrent panic attacks would benefit from referral to a psychotherapist for cognitive-behavioral therapy.

Scenario E

Diagnosis: mania

Diagnostic evaluation: Always screen for drugs of abuse. In a patient with no known history of bipolar disorder, look for a medical cause of these symptoms (eg, steroids, hyperthyroidism, neoplasm). Checking levels of prescribed mood stabilizers (valproic acid or lithium) can assess medication compliance.

Management: Psychiatric hospitalization will be necessary if this is a first manic episode or if the patient poses a risk to self or others, is grossly impaired by the symptoms, or is psychotic. Early offering of oral antipsychotics will help manage the patient's agitation while also treating the underlying process. Initiation of a mood stabilizer will likely take place on an inpatient unit.

HEMATOLOGIC DISORDERS

Recognition of Bleeding Disorders	911
Pathophysiology of Hemostasis	911
Laboratory Tests for Evaluation of Hemostasis	911
Bleeding Disorders and Their Clinical Parameters	914
Bleeding Disorders Associated with Platelet Function Problems and/or a Decreased Platelet Count	915
Platelet Disorders	915
Heparin-Induced Thrombocytopenia	918
Disseminated Intravascular Coagulation	920
Bleeding (Coagulation) Disorders Associated with an Increased Partial Thromboplastin Time and Normal Prothrombin Time	921
Hemophilia	921
von Willebrand Disease	922
Sickle Cell Disease	923
Anemia	926
General Approach	926
Clinical Presentation.....	926
Diagnostic Evaluation	927
Nonhereditary Anemia: Hemolytic Anemia	929
Acquired Nonhemolytic Anemias	929
Hereditary Anemia	931
Blood Transfusions	931
Transfusion of Specific Blood Products	931
Transfusion Methods.....	933
Complications	933
Reversal of Drug-Induced Anticoagulation	934
Reversal of Warfarin-Induced Coagulopathy	934
Reversal of Heparin and Low-Molecular-Weight Heparin	936
Reversal of Newer Anticoagulants	937
Reversal of Thrombolysis	937

HEMATOLOGIC DISORDERS: SELF-ASSESSMENT QUESTIONS

1. All of the following factors are vitamin K-dependent except:
 - (a) II
 - (b) VII
 - (c) IX
 - (d) XI
2. Isolated prolongation of the prothrombin time may be seen in each of the following patients except:
 - (a) Patients taking warfarin
 - (b) Patients with liver disease
 - (c) Patients with vitamin K deficiency
 - (d) Patients with hemophilia
3. The test that is most appropriate for evaluating the tissue factor pathway (extrinsic) is:
 - (a) Platelet aggregation studies
 - (b) Prothrombin time
 - (c) Thrombin time
 - (d) Partial thromboplastin time
4. All of the following laboratory findings are consistent with disseminated intravascular coagulation except:
 - (a) Increased fibrinogen level
 - (b) Decreased platelets
 - (c) Fragmented RBCs and anemia
 - (d) Increased fibrin split products
5. The laboratory test that distinguishes hemophilia A and B from von Willebrand disease is:
 - (a) Partial thromboplastin time
 - (b) Platelet function studies
 - (c) Prothrombin time
 - (d) Platelet count
6. Which of the following is effective in treating hemophilia A, hemophilia B, and von Willebrand disease (although not necessarily the therapy of choice)?
 - (a) Cryoprecipitate
 - (b) DDAVP
 - (c) Prothrombin complex
 - (d) Fresh frozen plasma

7. Which of the following labile clotting factors are most depleted in a stored unit of blood?
- (a) Factors VIII and I
 - (b) Factors VIII and V
 - (c) Factors VII and V
 - (d) Factors IX and XI
8. All of the following statements regarding cryoprecipitate are true except:
- (a) It is effective in treating all types of von Willebrand disease.
 - (b) It contains large quantities of factors I, VIII, and IX.
 - (c) Use of this product is associated with risk of transmission of the AIDS and hepatitis viruses.
 - (d) Although not the therapy of choice, it is effective in treatment of hemophilia A.
9. Which of the following statements about aplastic crisis in sickle cell patients is inaccurate?
- (a) It is frequently precipitated by an infection.
 - (b) Patients complain of abdominal pain, and physical examination reveals splenomegaly.
 - (c) The diagnosis is suggested by the presence of a decreased reticulocyte count and a low hemoglobin.
 - (d) Patients are usually shocky and require oxygen, folic acid, fluid resuscitation, and transfusion.
10. The most accurate test for evaluating the adequacy of anticoagulation in a patient on chronic warfarin therapy is:
- (a) Prothrombin time
 - (b) Partial thromboplastin time
 - (c) INR
 - (d) The prothrombin time and INR are of equal accuracy in such a patient and can be used interchangeably.
11. A 7-year-old white boy is brought in from summer camp for evaluation of a head injury. The counselor accompanying him reports that the boy was accidentally struck in the head with a bat. The injury occurred about 30 minutes ago and according to the counselor, the boy now seems somewhat more lethargic. The counselor also states that the child has hemophilia A. Examination reveals a frontal hematoma and a resolving hemarthrosis of the left knee. You decide to immediately administer factor VIII concentrate to this child in whom you suspect CNS bleeding. The most appropriate dosage of factor VIII concentrate is:
- (a) 18 units/kg
 - (b) 26 units/kg
 - (c) 50 units/kg
 - (d) Cannot be determined from the information given

12. A 40-year-old woman presents with easy bruising, gingival bleeding when she brushes her teeth, and menorrhagia. Examination reveals multiple purpuric lesions that are particularly prevalent on the lower extremities, but there is no active bleeding. Laboratory findings include a platelet count of 25,000/mm³, a hemoglobin of 12.8 g/dL, a BUN of 15 mg/dL, a creatinine of 0.8, and a normal prothrombin time (INR)/partial thromboplastin time. The most appropriate initial therapy is:
- (a) Platelet transfusion
 - (b) Plasmapheresis
 - (c) Corticosteroids
 - (d) Immediate splenectomy
13. A 50-year-old man presents with profuse rectal bleeding. He is currently on warfarin, and his INR is 5.2. His hemoglobin is 10.2 g/dL. The most appropriate initial therapy is:
- (a) Platelet transfusion
 - (b) IV vitamin K
 - (c) DDAVP
 - (d) Cryoprecipitate
14. Which of the following statements about anticoagulation reversal is false?
- (a) Owing to the risk of anaphylaxis, vitamin K should not be given IV.
 - (b) Prothrombin complex concentrate (PCC) is indicated for the reversal of warfarin in patients with an increased INR who are bleeding.
 - (c) Although not FDA approved, factor VII inhibitor bypassing activity (FEIBA) has been shown in small studies to reverse the anticoagulant effects of dabigatran and rivaroxaban.
 - (d) Protamine can partially reverse the effects of low-molecular-weight heparin (LMWH).
15. A patient presents with progressive fatigue and shortness of breath. Laboratory studies show a hemoglobin of 8 g/dL, WBC count of $8.3 \times 10^9/L$, and a platelet count of 210/mm³. Her reticulocyte count is increased, and her serum haptoglobin is low. She has a total bilirubin of 4 and a direct bilirubin of 1.2. What is the most likely cause of the anemia?
- (a) Hemolysis
 - (b) Iron deficiency
 - (c) Thrombotic thrombocytopenia purpura
 - (d) Bone marrow failure

ANSWERS

- | | | | | |
|------|------|------|-------|-------|
| 1. d | 4. a | 7. b | 10. c | 13. b |
| 2. d | 5. b | 8. b | 11. c | 14. a |
| 3. b | 6. d | 9. b | 12. c | 15. a |

Use the pre-chapter multiple choice question worksheet (page xx) to record and determine the percentage of correct answers for this chapter.

I. RECOGNITION OF BLEEDING DISORDERS

A. Pathophysiology of hemostasis

1. Hemostasis is a balance between excessive bleeding and thrombosis. It occurs when four components (listed below) interact synergistically with one another. If a component is not functioning normally, or it fails to interact appropriately with another component, excessive bleeding or clotting occurs.
2. Components of hemostasis
 - a. Vascular integrity: primary
 - b. Platelet function: primary
 - c. Coagulation factors: secondary
 - d. Fibrinolysis
3. Bleeding disorders result from a defect in one or more of the components of hemostasis.
 - a. Inflammation of blood vessel walls, or a defect in the vessel supportive connective tissue, causes increased aggregation of platelets at the vessel site but does not affect the total number of circulating platelets. Bleeding, therefore, is localized to the vessel site.
 - b. Platelet function depends on an adequate number of circulating platelets, as well as normally functioning platelets. Bleeding results when either the count is low or the function is abnormal.
 - c. Coagulation factors are activated by substances in injured tissue that set off a series of steps that terminate in the formation of a fibrin clot. If there is an inadequate amount of one or more factors, or if a factor is abnormal, this series of steps is disrupted, thus preventing formation of a fibrin clot.
 - d. Fibrinolysis normally results in dissolution of the fibrin clot as the vascular defect is repaired. Excessive fibrinolysis causes bleeding by functionally altering coagulation factors or by inducing excessive consumption of coagulation factors.

B. Laboratory tests for evaluation of hemostasis

1. Platelet count

- a. Normal range: 150,000-400,000/mm³
- b. Decreased in any condition that causes destruction, sequestration, or decreased production of platelets:
 - (1) Idiopathic (autoimmune) thrombocytopenic purpura (ITP)
 - (2) Thrombotic thrombocytopenic purpura (TTP)
 - (3) Leukemia
 - (4) Bone marrow suppression/replacement
 - (5) Drug/toxic effect
 - (6) Hypersplenism
 - (7) Disseminated intravascular coagulation (DIC)
 - (8) HIV infection
 - (9) Sepsis (especially gram-negative sepsis)
 - (10) Aplastic/hypoplastic anemia
- c. Post-traumatic bleeding occurs if the platelet count falls below 50,000/mm³; spontaneous, life-threatening hemorrhage can occur when the platelet count falls below 10,000-20,000/mm³.

- d. As many as 50% of patients with unexplained thrombocytosis (\uparrow platelet count) have an underlying malignancy.
2. Testing of platelet function
 - a. Bleeding time is no longer used clinically and is mentioned here only for historical purposes. It measures the time between skin incision and cessation of bleeding. Duration of this type of bleeding depends on the integrity of primary hemostasis (platelets, von Willebrand factor) and is independent of coagulation.
 - b. Platelet function testing is not routinely used and has limited utility in the emergency department. (However, the emergency physician may come across these tests and their results and should at least be familiar with the concept.) It has replaced bleeding time as the test of choice for evaluating platelet activity. Platelet aggregation studies can confirm the inhibitory effects of ASA, thienopyridines (ticlopidine, clopidogrel), β -lactam antibiotics, and other factors on platelet function.
 - c. Platelet function tests measure platelet aggregation after exposure to a panel of agonists, which may include ADP, epinephrine, collagen, arachidonic acid, ristocetin, and thrombin. The pattern obtained allows for the diagnosis and classification of the platelet defect.
3. Prothrombin time (PT)
 - a. Tests the factors of the tissue factor (extrinsic) pathway (primarily factor VII) and the common pathway (X, V, II, and I).
 - b. Normal control values are 10–12 seconds.
 - c. Prolongation of PT ≥ 2 seconds is considered significant.
 - d. Increased PT (with a normal PTT) results from a deficiency of factor VII. Causes of factor VII deficiency are:
 - (1) Hereditary (rare)
 - (2) Acquired secondary to:
 - (a) Vitamin K deficiency
 - (b) Warfarin use
 - (c) Liver disease
 - (3) Although the PT is very useful in evaluating patients with hemostatic abnormalities, it should not be used to monitor the adequacy of anticoagulation with warfarin. The response of the PT to a dose of warfarin is very dependent on the thromboplastin used in the assay, which varies from laboratory to laboratory. Thus, reliance on the PT alone in these patients can result in excessive or suboptimal anticoagulation and an increased risk of bleeding complications. For this reason, it is now recommended that the international normalized ratio (INR) be used to monitor these patients.
4. INR
 - a. The INR is the PT ratio that would be obtained if the World Health Organization's reference thromboplastin had been used.
 - b. It is the test of choice for monitoring the adequacy of anticoagulation in patients taking warfarin.
 - c. An INR of 1 is normal.
 - d. An INR of 2–3 is considered to be therapeutic for most patients, while an INR of 2.5–3.5 is recommended for patients with mechanical prosthetic valves and recurrent embolization.

5. **Partial thromboplastin time (PTT)**
 - a. Essentially tests all of the factors except VII and XIII but is used primarily to evaluate the factors of the contact activation (intrinsic) pathway; it tests components of both the contact activation (intrinsic) and common pathways.
 - b. Normal control values range from 25 to 35 seconds.
 - c. A prolongation of 8–10 seconds above normal is considered significant.
 - d. Increased PTT (with a normal PT) is seen with:
 - (1) Factor VIII, IX, or XI deficiency (primarily)
 - (2) Factor XII deficiency (asymptomatic)
 - (3) Heparin therapy
 - e. It is common to see the PTT reported as "aPTT," which stands for activated partial thromboplastin time, called this because an "activating" agent (kaolin) was added to the plasma before the test was run.
 - f. When laboratory tests reveal an increased PT and increased PTT, this indicates an abnormality primarily in the common pathway (associated abnormalities of the intrinsic and extrinsic pathways may also be present).
 - (1) Heparin overdose (factors X and II are primary sites of action)
 - (2) Warfarin overdose (inhibits factors II, VII, IX, and X)
 - (3) Vitamin K deficiency and moderate to severe liver disease (inhibit production of factors II, VII, IX, and X)
 - (4) DIC (consumes factors I, V, VIII, and XIII)
 - (5) Functional abnormality or deficiency of fibrinogen (factor I)
 - (6) Massive transfusion of stored blood (deficient in factors V, VIII)
 - (7) Thrombolytic therapy
6. **Thrombin time**
 - a. Tests the ability of fibrinogen (factor I) to convert into a fibrin clot. This is a useful screening test for both qualitative and quantitative abnormalities of fibrinogen.
 - b. Increased thrombin time is seen with:
 - (1) Hypofibrinogenemia
 - (2) Myeloma, macroglobulinemia (interferes with fibrin polymerization)
 - (3) Heparin therapy (inhibits fibrinogen formation)
 - (4) DIC (consumes fibrinogen)
 - (5) Liver disease
 - (6) Uremia
7. **Fibrinogen degradation products test for specific degradation products of fibrin.** Increased levels are seen with consumption coagulopathies (eg, DIC) and thrombolytic agent therapy.

C. Bleeding disorders and their clinical parameters

Table 37: Clinical Parameters of Hemostatic Disorders

Hemostatic Disorder	Clinical Findings	Platelet Function Studies	Platelets	PT	PTT	Fibrinogen	Clinical Disorder
Primary hemostasis: platelet mediated							
Thrombocytopenia	Purpura; petechia; epistaxis; GI, GU, and menstrual bleeding	Normal	↓	Normal	Normal	Normal	ITP, TTP, hemolytic uremic syndrome, heparin-induced thrombocytopenia (abnormal PTT if still on heparin), others
Platelet dysfunction	Purpura; petechia; epistaxis; GI, GU, and menstrual bleeding	Abnormal	Normal	Normal	Normal	Normal	ASA, clopidogrel, ticlopidine, inherited disorders, others
von Willebrand disease	Purpura; petechia; epistaxis; GI, GU, and menstrual bleeding; hemarthrosis and muscular bleeding ^a	Abnormal	Normal	Normal	Normal to ↑ ^a	Normal	von Willebrand disease
Secondary hemostasis: coagulation							
Warfarin therapy		Normal	Normal	↑	Normal to ↑ ^b	Normal	Warfarin therapy, rat poison (brodifacoum, others)
Heparin		Normal	Normal	Normal	↑	Normal	Heparin therapy
Low-molecular-weight heparin (LMWH)	Intramuscular, intracranial, GI, postoperative, and traumatic bleeding	Normal	Normal	Normal	Normal to mildly ↑ ^c	Normal	Enoxaparin, dalteparin, fondaparinux, tinzaparin
Hemophilia A		Normal	Normal	Normal	↑	Normal	Factor VIII levels decreased
Hemophilia B (Christmas disease)		Normal	Normal	Normal	↑	Normal	Factor IX levels decreased
Combined disorders of platelets and coagulation							
Disseminated intravascular coagulation	Thrombosis, microangiopathic hemolytic anemia, bleeding (purpura; petechial; epistaxis; GI, GU, menstrual, intramuscular, intracranial, postoperative, and traumatic bleeding)		↑	↑	↑	↑ ^d	Sepsis, trauma, burns, acute promyelocytic leukemia, amniotic fluid embolism, placental abruption, snake bites (pit viper), liver disease

^a In type 3 and type 2N (abnormal binding of von Willibrand factor to factor VIII, resembling hemophilia A)

^b If INR/PT is sufficiently prolonged by warfarin, PTT will also be prolonged, owing to warfarin's effect on the common pathway.

^c LMWH is monitored using antiXa levels.

^d Fibrin split products and d-dimers are increased as the result of concomitant thrombolysis during DIC.

II. BLEEDING DISORDERS ASSOCIATED WITH PLATELET FUNCTION PROBLEMS AND/OR A DECREASED PLATELET COUNT

A. Platelet disorders

1. Thrombocytopenia (the most common platelet abnormality)

a. Etiology

- (1) Decreased bone marrow production
 - (a) Malignant infiltration of bone marrow
 - (b) Myelofibrosis
 - (c) Aplastic anemia
 - (d) Drug suppression (ethanol, thiazide diuretics, chemotherapeutic agents)
 - (e) Radiation therapy
 - (f) Viral infections
 - (g) Vitamin B₁₂/folate deficiency
- (2) Hypersplenism (cirrhosis, uremia, sequestration)
- (3) Increased platelet destruction
 - (a) ITP
 - (b) TTP
 - (c) Drug-related destruction (eg, penicillin, sulfonamides, quinine, furosemide, heparin, ASA, gold salts, cimetidine, digoxin)
 - (d) Hemolysis, elevated liver enzymes, and low platelet count (HELLP) syndrome
 - (e) Sepsis
 - (f) Viral infection: HIV, mumps, varicella, Epstein-Barr virus
 - (g) Collagen vascular diseases (especially lupus erythematosus)
 - (h) Hemolytic uremic syndrome
 - (i) Post-transfusion (middle-aged woman previously sensitized to the P1A1 antigen during pregnancy who received a transfusion 1 week ago)

b. Classic clinical scenario

- (1) A patient (usually a woman) presents with easy bruising, epistaxis, gingival bleeding, hematuria, menorrhagia, and/or GI bleeding. The bleeding may be spontaneous or secondary to trauma and is usually controlled by local pressure. When secondary to trauma, the bleeding has immediate onset. In some cases, the patient may also relate that she was recently started on a new medication.
- (2) Examination reveals multiple petechiae and purpura on the mucous membranes and skin, particularly on the dependent parts of the body.
- (3) Laboratory findings include a low platelet count and a normal PT(INR)/PTT.

c. Indications for platelet transfusion (6 units of random donor platelet concentrate increases the platelet count by 50,000/mm³)

- (1) Platelet count <20,000/mm³
- (2) Life-threatening bleeding with evidence of platelet dysfunction or consumption

- (3) Patients with autoimmune disorders such as ITP can tolerate platelet levels as low as $1,000/\text{mm}^3$ without being transfused; also, if the problem is secondary to TTP, platelets are contraindicated.

d. Admission criteria

- (1) All newly diagnosed patients with a serious platelet disorder
- (2) All patients with persistent bleeding unresponsive to outpatient therapy (regardless of platelet count)

2. ITP

a. Acute ITP (most common hemorrhagic disease in children)

(1) Clinical presentation

- (a) Most often affects children 2–6 years old and frequently follows a viral infection
- (b) This is a self-limited disorder that usually resolves spontaneously within a few weeks to months.

(2) Diagnostic evaluation

- (a) Diagnosis is based on history (medications), physical examination (platelet-type bleeding), and peripheral smear/platelet count (CBC).
- (b) Adults usually present with platelet counts $<10,000/\text{mm}^3$ and therefore require therapy.

(3) Emergency treatment for severe bleeding (platelets $<50,000/\text{mm}^3$) or a platelet count $<20,000/\text{mm}^3$ without bleeding

- (a) Primarily supportive; RBC transfusion is indicated for anemia or hypotension.
- (b) Steroids: prednisone orally 1–1.5 mg/kg/day (newer studies have demonstrated efficacy of high-dose dexamethasone as initial treatment [40 mg/day orally \times 4 days])
- (c) Intravenous immunoglobulin (IVIG) for platelet counts $<5,000/\text{mm}^3$ in patients who have completed a several-day course of steroids
- (d) Platelet transfusions in consultation with a hematologist may be indicated for life-threatening hemorrhage. Transfused platelets are rapidly destroyed.

(4) Pediatric considerations

- (a) Treatment of children is controversial because of favorable outcomes without therapy (in $>70\%$, acute ITP resolves within 6 months). Concern for intracranial hemorrhage drives the physician to treat these children. Most ICHs occur within 4 weeks of presentation with platelet counts $<20,000/\text{mm}^3$.
- (b) Because there is no proof that therapy prevents ICH, current recommendations are for a pediatric hematologist to manage these patients on an outpatient basis. Parents are advised to limit the child's physical activity to prevent further trauma and avoid medications that may aggravate bleeding (ASA, ibuprofen, etc).

b. Chronic refractory ITP

- (1) Usually affects women 20–40 years old and is often insidious in onset. Spontaneous remission is uncommon, and an underlying autoimmune, collagen vascular, or malignant disease is present in many of these patients.

(2) Treatment

- (a) Initial therapy is with prednisone.

- (b) Steroid-refractory patients and those with active bleeding may subsequently require one or more of the following:
 - i. Splenectomy
 - ii. Intravenous immunoglobulin (IVIG)
 - iii. Immunosuppressive therapy (azathioprine, cyclophosphamide)
 - iv. Rituximab
 - v. Platelet transfusion (for life-threatening hemorrhage only)
 - vi. Thrombopoietin agonists (eg, romiplostim, eltrombopag)
- (c) The goal of therapy is a platelet count of 30,000–50,000/mm³.
- c. ITP during pregnancy
 - (1) In women with ITP, platelet counts may decrease during pregnancy.
 - (2) Platelet counts should be maintained >30,000/mm³ during pregnancy (and >50,000/mm³ near term) to minimize the need for platelet transfusion.
- 3. TTP
 - a. TTP is a subset of a larger group of pathologies called thrombotic microangiopathies.
 - b. Exists on a spectrum with and shares some features with hemolytic uremic syndrome
 - c. More common in women
 - d. The underlying pathophysiology is reasonably well understood.
 - e. Pathologically results from deposition of microthrombi in vessel lumen
 - f. Mortality is 80% if untreated.
 - g. Early diagnosis is a key factor in survival.
 - h. Clinical presentation
 - (1) It is rare to see all of these findings in TTP, especially if the patient presents early in the course of the disease. Later in the course, more findings may be present.
 - (a) Fever: least common finding in TTP
 - (b) Anemia: microangiopathic hemolytic anemia
 - (c) Thrombocytopenia
 - (d) Renal abnormalities: less common in TTP and more common in hemolytic uremic syndrome
 - (e) Neurologic abnormalities: found in two-thirds of patients with TTP
 - (2) Most patients in this era do not have all five features, because it is now treated before progression to its natural end; untreated, the mortality is ~80%.
 - (3) All that is needed for diagnosis is thrombocytopenia and microangiopathic hemolytic anemia (produces schistocytes)
 - (4) Neurologic symptoms in two-thirds of cases; global or focal via CNS thrombosis
 - (5) Renal failure is more common in hemolytic uremic syndrome but can also happen in TTP.
 - (6) Fevers are less common.
 - (7) High mortality rate if untreated
 - (8) An increased lactate dehydrogenase and a decreased haptoglobin are usually seen in TTP and are markers of hemolysis.

i. TTP versus DIC

(1) DIC is a "consumptive coagulopathy."

- (a) Low platelets
- (b) Low coagulation factors (high PTT and INR)
- (c) Low fibrinogen
- (d) Microangiopathic hemolytic anemia and fibrin plugs

(2) TTP is a platelet problem

- (a) Low platelets
- (b) Normal coagulation factors (normal PTT and INR)
- (c) Normal fibrin
- (d) Microangiopathic hemolytic anemia and platelet plugs

j. Treatment

- (1) Begin steroids: methylprednisolone 125 mg
- (2) Dialysis may be needed if renal failure.
- (3) Plasma exchange transfusion is the key.
- (4) Give fresh frozen plasma (FFP) if plasma exchange will be delayed for any reason (extended transfer times, equipment issues, etc).
- (5) Avoid platelet transfusions.

B. Heparin-induced thrombocytopenia (HIT)

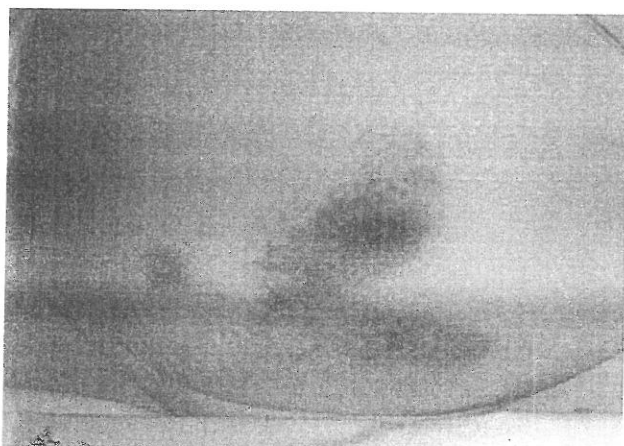
1. Epidemiology

- a. Up to 5% of heparinized patients
- b. Develops in 5–10 days
- c. Can develop earlier if the patient has had prior HIT episodes
- d. Late presentation (>10 days after heparin) is also possible but less common.
- e. Occurs as the result of an IgG antibody that forms against heparin when it is bound to platelet factor 4
- f. HIT can develop after heparin doses as small as those used for repetitive heparin flushes.
- g. HIT can develop in patients who are heparinized repeatedly for dialysis.
- h. Regular heparin > LMWH
- i. Surgical > medical > obstetrics patients
- j. Female > male

2. Clinical presentation (4 T's)

- a. Thrombocytopenia ($<150 \times 10^9/L$): A mild or moderate thrombocytopenia is seen. Median platelet count nadir is $60 \times 10^9/L$ (range from 15 to $150 \times 10^9/L$ in 90% of patients). A more practical definition includes a platelet count drop of 50% from baseline. Postoperative patients generally have increased platelet counts. They may be on heparin in the hospital or may be on SC heparin or LMWH after discharge. If a postoperative platelet count is $350 \times 10^9/L$ and the patient presents 3 days after discharge with a platelet count of $175 \times 10^9/L$, this represents a 50% drop in platelet count, but it does not meet the traditional definition of thrombocytopenia. In the right clinical scenario, HIT should be considered in this type of patient.
- b. Thrombosis: arterial or venous
- c. Timing (in relation to heparin): generally 5–10 days after starting heparin

- d. No other explanation
 - e. Necrotic skin lesion at heparin/LMWH injection site
 - f. Warfarin skin necrosis in the form of limb gangrene
 - g. Pulmonary embolism/deep-venous thrombosis despite thrombocytopenia
3. Treatment: The 3 Do's
- a. Stop heparin or LMWH!
 - b. Start alternative anticoagulant. This should be a direct thrombin (factor IIa) inhibitor. Don't delay! These drugs include argatroban, lepirudin, and bivalirudin.
 - c. Reverse warfarin. If INR is increased, warfarin reversal should be done with vitamin K 10 mg orally. There is no need to use other reversal agents such as FFP, prothrombin complex concentrates (eg, nonactivated factor IX complex), or recombinant coagulation factor VIIa, because there is no active bleeding and reversal is not time dependent. (Warfarin reversal seems counterintuitive; why would you want to reverse anticoagulation in someone who is now hypercoagulable? Disturbance in procoagulant-anticoagulant balance is the reason for the need to reverse warfarin. Warfarin has two major effects. First, it causes an immediate decrease in protein C-mediated anticoagulation. This means that warfarin initially has a procoagulant effect. After a few days of use, warfarin also inhibits the production of active forms of factors II, VII, IX, and X. This is its anticoagulant effect. After a few days, the net balance of these two effects is to cause anticoagulation. When protein C levels are decreased by warfarin and HIT antibodies are present [procoagulants], the balance shifts toward the formation of clots.)
4. Treatment: The 2 Don'ts
- a. Avoid warfarin: reverse with vitamin K if INR increased.
 - b. Avoid platelet transfusions: may worsen the hypercoagulability.
5. Diagnostic evaluation
- a. Investigate for lower-limb deep-venous thrombosis (duplex ultrasonography).
 - b. Test for HIT antibodies. This looks for IgG antibodies to the heparin-platelet factor 4 complex. This is not a useful test for the emergency physician. It is worth sending the test for the purpose of confirming the diagnosis for the inpatient physicians. The test will take a while to complete and will probably be a "send out" test for most emergency departments.



Courtesy of Colin Kaide, MD

Bleeding at LMWH injection site with HIT



Courtesy of Colin Kaide, MD
Clotting in extremity with HIT

C. Disseminated intravascular coagulation (DIC)

1. Definition: life-threatening bleeding disorder resulting from loss of platelets and coagulation factors, fibrinolysis, small vessel occlusion, and RBC destruction from fibrin deposition
2. Clinical presentation
 - a. Patients usually present with diffuse bleeding from multiple sites (skin, mucous membranes, and viscera) that is not controlled by local measures.
 - b. At times, acrocyanosis, thrombosis, and pregangrenous changes of fingers, toes, genitalia, and nose are presenting symptoms (purpura fulminans).
3. Etiology
 - a. Trauma (including burns, crush injuries, and brain trauma)
 - b. Pregnancy complications (placental abruption, amniotic fluid emboli, fetal death in utero)
 - c. Sepsis and a host of infectious diseases
 - d. Transfusion and drug reactions
 - e. Carcinoma and acute leukemias
 - f. Liver disease
 - g. Snake bites
 - h. Prosthetic devices (especially peritoneovenous shunts)
 - i. Heat stroke
4. Diagnostic evaluation
 - a. Clinical and laboratory triads
 - (1) Purpura (petechiae) → decreased platelets
 - (2) A bleeding tendency → increased PT(NR)/PTT
 - (3) Signs of organ injury → decreased fibrinogen
 - b. Additional laboratory findings
 - (1) ↑ Fibrin split products (the earliest sign) and ↑ D-dimer (most specific sign)
 - (2) ↑ Thrombin time
 - (3) Fragmented RBCs (schistocytes): evidence of microangiopathic hemolytic anemia

5. Treatment

- a. Primary therapy is directed toward the underlying cause when it is easily identifiable (sepsis, pregnancy, drug or transfusion therapy) and correction of hemodynamic instability (IV fluids and packed RBCs).
- b. Specific therapy is based on the clinical presentation.
 - (1) If hemorrhage predominates the clinical picture, then blood component therapy is indicated. (All three should be given.)
 - (a) Platelets
 - (b) FFP (coagulation factors and fibrinogen)
 - (c) Cryoprecipitate (factors I and VIII)
 - (2) If fibrin deposition and thrombosis predominate the clinical picture, then low-dose IV heparin is indicated. Disease states in which this occurs include:
 - (a) Gram-negative sepsis in pregnancy
 - (b) Retained dead fetus
 - (c) Chronic DIC (eg, carcinoma)
 - (d) Purpura fulminans
 - (3) Antithrombin III concentrates may also be used to treat DIC, either alone or in conjunction with heparin. Antithrombin III is an endogenous inhibitor of coagulation that binds to and inactivates thrombin and several other factors.

III. BLEEDING (COAGULATION) DISORDERS ASSOCIATED WITH AN INCREASED PARTIAL THROMBOPLASTIN TIME (PTT) AND NORMAL PROTHROMBIN TIME (PT)

A. Hemophilia

1. Characteristics of both hemophilia A and hemophilia B
 - a. Bleeding history
 - (1) Delayed and protracted bleeding after mild trauma or dental extractions
 - (2) Spontaneous hematuria
 - (3) Hemarthrosis and muscle hematomas (~90% of all bleeding)
 - (4) Intracranial hemorrhages (a major cause of death in patients with hemophilia A)
 - b. Diagnostic evaluation
 - (1) Prolonged PTT
 - (2) Normal PT and platelets
2. Hemophilia A (classic hemophilia)
 - a. A sex-linked recessive disorder due to a deficiency of factor VIII:C
 - b. In addition to a prolonged PTT, assay of factor VIII:C is abnormal.
 - c. Treatment
 - (1) Factor VIII concentrate: the treatment of choice for those with moderate to severe disease. The products that are now available are all free of the risk of transmission of AIDS and hepatitis (types B and C) and include:

- (a) Heat-treated factor VIII concentrate (hepatitis A and parvovirus transmission is still possible)
- (b) Monoclonal antibody purified factor VIII concentrate
- (c) Recombinant factor VIII concentrate (an ultrapure product)
- (d) Dosing: primarily determined by the type/location of bleeding
 - i. Mild bleeding (hematuria, early hemarthrosis, deep laceration) → 12.5 units/kg
 - ii. Moderate bleeding (oral lacerations, dental extraction or minor surgery, late hemarthrosis) → 25 units/kg
 - iii. Severe bleeding (CNS, GI, intra-abdominal or retroperitoneal bleeding; major trauma or surgery, documented or potentially serious head injury) → 50 units/kg
 - iv. Each unit of factor VIII infused per kg of body weight increases the plasma factor VIII level by 2%. In a suspected bleeding emergency, a hemophiliac patient's present level of factor VIII is assumed to be zero unless previously known.
- (2) Cryoprecipitate carries a risk of hepatitis and AIDS viruses as well as of allergic reactions and, for this reason, its use has declined; it is no longer considered first-line therapy. (One bag equals 80–100 units of factor VIII).
- (3) Desmopressin (DDAVP) is effective in increasing the levels of factor VIII in patients with mild hemophilia and is the treatment of choice for these patients; dosage is 0.3 mcg/kg. There is no risk of infectious disease transmission.
- (4) FFP: volume constraints generally limit its use to patients with mild hemophilia. Infectious disease transmission can occur. (1 unit of FFP raises the factor level only 3%–5%)
- 3. Hemophilia B (Christmas disease)
 - a. A sex-linked recessive disorder caused by a deficiency of factor IX
 - b. In addition to a prolonged PTT, assay of factor IX is abnormal.
 - c. Treatment
 - (1) Factor IX concentrate is currently the treatment of choice for patients with hemophilia B. The monoclonal concentrates are free from the risk of transmission of hepatitis and AIDS.
 - (2) Prothrombin complex concentrates contain factors II, VII, IX, and X, which are also effective but have thrombogenic adverse effects and may transmit hepatitis.
 - (3) FFP is effective too, but carries the risk of infectious disease transmission.
- B. von Willebrand disease (vWD)
 - 1. This is an autosomal (usually dominant) disorder affecting both males and females.
 - 2. vWD is the most common genetic bleeding disorder. There are three major types:
 - a. Type I (most common): decreased von Willebrand factor (vWF)
 - b. Type II: abnormal or dysfunctional vWF
 - c. Type III (rare): almost no vWF if present (serious disease)
 - 3. Depending on the specific type of vWD present, these patients have a deficiency or abnormality of one or more of the following:
 - a. vWF: promotes platelet adhesion; this is the primary deficiency found in vWD.
 - b. vWF:Ag
 - c. Factor VIII:C: protected by vWF

4. Clinical presentation
 - a. Mucocutaneous bleeding (especially epistaxis, menorrhagia, and GI bleeding) predominates; hemarthroses, therefore, are rare.
 - b. Bleeding episodes are usually milder than with hemophilia (particularly hemophilia A, which is typically associated with the most severe bleeding).
5. Laboratory studies
 - a. Increased PTT
 - b. Abnormal platelet function studies
 - c. Normal platelet count and PT
 - d. Abnormal assay of one or more of the following: vWF, vWF:Ag, or factor VIII:C
6. Management is determined by the type of vWD present.
 - a. DDAVP: use is restricted to patients with mild to moderate type I vWD; it is the treatment of choice in these patients and has no risk of infectious disease transmission.
 - b. Factor VIII concentrates containing large amounts of vWF (Humate-P and Koate-HS) are currently the treatment of choice for patients with type II and type III disease. Both products have been treated to eliminate the risk of transmission of infectious diseases.
 - c. Cryoprecipitate (usual dose = 10 units/kg) is effective for all types of vWD but carries risk of transmission of hepatitis and HIV infection.
 - d. FFP is also effective for all types of vWD but carries risk of transmission of infectious diseases. FFP may be used to treat all forms of hemophilia (hemophilia A and B and vWD), because it contains all factors. The major limitation to its use is volume overload; compared with factor VIII concentrate, factor IX concentrate, prothrombin complex, and cryoprecipitate, the concentration of factors in FFP (1 unit/mL) is low. Therefore, excessively large volumes would be required to treat patients with moderate or severe disease. FFP is most appropriate for patients in whom the exact nature of their hemophilia is unknown.

IV. SICKLE CELL DISEASE

- A. A genetic disorder found predominantly in people of African ancestry that is due to an abnormal hemoglobin molecule (hemoglobin S). The normal hemoglobin molecule is hemoglobin A. Hemoglobin S induces red cell deformation (sickling). These sickled cells have two properties that account for their pathologic manifestations:
1. Sickled RBCs are unable to pass freely through the systemic microvasculature. This leads to thrombosis (ischemia, infarction).
 2. Sickled RBCs hemolyze more easily than normal RBCs. This leads to chronic hemolytic anemia and increased susceptibility to infection.

Table 38: Sickle Cell Hemoglobin (Hb)

Phenotype	Genotype	Hemoglobin	Abnormality
Sickle trait	Hb A/S	$\alpha_2 \beta_1 \beta_1$	Valine for Glu at 6th position
Sickle cell disease	Hb S	$\alpha_2 \beta_2$	Valine for Glu at 6th position
Sickle-thalassemia	Hb S/A-thal*	$\alpha_2 \beta_2/\alpha_2 \beta_2$ -thal	thal=decreased production of Hb A
Sickle cell disease	Hb S + Hb C**	$\alpha_2 \beta_2/\alpha_2 \beta_2$	Lysine for Glu at 6th position

* In thalassemias, Hb A is produced in decreased amounts.

** In sickle cell disease, both forms of hemoglobin exist (Hb S and Hb C).

B. Sick cell trait (heterozygous hemoglobin AS)

1. When hemoglobin S is inherited from one parent and hemoglobin A from the other, RBCs will contain both hemoglobin A and hemoglobin S (<50% of the cells will be sickled).
2. Clinical presentation
 - a. Spontaneous bleeding (hematuria, hyphema)
 - b. Hyposthenuria (impaired ability to concentrate urine)
 - c. Vaso-occlusive crises (are rare and occur only under conditions of extreme hypoxia)

C. Sick cell anemia (homozygous hemoglobin SS)

1. When hemoglobin S is inherited from both parents, most of the RBCs will contain only hemoglobin S (75%–95% of the cells will be sickled).
2. Clinical presentation in children
 - a. Unexplained gnawing bone pain
 - b. Left upper quadrant pain (splenic infarctions)
 - c. Right upper quadrant pain (gallstones)
 - d. Severe hemolytic anemia
 - e. Jaundice
 - f. Painful swelling of the hands and/or feet (dactylitis) may be the presenting complaint in children <2 years old.
 - g. Increased susceptibility to infections (sepsis, meningitis, pneumonia)
 - (1) Encapsulated organisms (*Streptococcus pneumoniae*, *Haemophilus influenzae*) are the most common pathogens.
 - (2) *Salmonella* osteomyelitis is also common.
3. Diagnostic evaluation
 - a. CBC: ↓ hemoglobin + ↑ WBCs + ↑ platelets; sickled RBCs on peripheral smear (RBCs not stippled)
 - b. Increased reticulocyte count
 - c. Hemoglobin electrophoresis confirms hemoglobin SS (and AS)

D. Sick cell crises

1. Vaso-occlusive (thrombotic) crisis
 - a. Severe unremitting pain in the back, abdomen, chest, or long bones
 - b. Diagnostic evaluation
 - (1) CBC to exclude hemolytic sequestration and aplastic crises
 - (2) Reticulocyte count to exclude aplastic crisis
 - (3) Urinalysis, chest radiograph, and cultures as indicated to exclude infection
 - c. Treatment: analgesics, hydration, folic acid supplementation, and antibiotics (if indicated)
2. Hemolytic crisis
 - a. Clinical presentation: patient appears very ill and is jaundiced.
 - b. Diagnostic evaluation: as above and type, screen, and cross
 - c. Treatment: as above and support shock (10–20 mL/kg IV of normal saline or lactated Ringer's); blood transfusion as needed.
3. Sequestration crisis (most common precipitating cause of acute anemia)
 - a. Seen in young children (6 months to 6 years old)

- b. **Clinical presentation:** patient presents with abdominal pain and distention, pallor, shock, and splenomegaly.
 - c. **Diagnostic evaluation**
 - (1) As above
 - (2) Hemoglobin very low (may be as low as 1–2 g) and pancytopenia
 - d. **Treatment:** as above and consultation with a pediatric surgeon for possible splenectomy
- 4. **Aplastic crisis**
 - a. Usually precipitated by an infection (most often human B19 parvovirus) that suppresses erythropoiesis; folate deficiency is rarely the underlying cause.
 - b. **Clinical presentation:** patient is pale, lethargic, and in shock.
 - c. **Diagnostic evaluation**
 - (1) As above and appropriate cultures
 - (2) Decreased reticulocyte count (secondary to severe bone marrow depression) is pathognomonic and associated with a very low hemoglobin.
 - d. **Treatment**
 - (1) Support shock and transfuse blood.
 - (2) Administer oxygen, folic acid, and antibiotics as indicated.
- E. **Other potential complications and sequelae of sickle cell disease**
 - 1. Infections (most common cause of death)
 - a. Sepsis, meningitis, pneumonia, and osteomyelitis occur with increased frequency due to functional asplenia, poorly migrating neutrophils, and decreased opsonin activity.
 - b. Children (particularly those <2 years old) with unexplained high fever (>103°F [39.4°C]) and a WBC count >20,000/mm³ should be promptly treated with broad-spectrum IV antibiotics, and cultures should be done.
 - 2. Priapism
 - a. Definition: a painful, sustained, pathologic erection
 - b. Occurs in 10%–40% of men and can result in impotence
 - c. Treatment
 - (1) Obtain immediate urologic consult and administer terbutaline 0.25–0.5 mg SC.
 - (2) Subsequent measures
 - (a) Hydration
 - (b) Transfusion of packed RBCs
 - (c) Hyperbaric oxygen
 - (d) Analgesia (appropriate for treatment of pain but ineffective in relieving priapism)
 - 3. **Acute chest syndrome**
 - a. Most commonly affects older children, adolescents, and adults
 - b. Patients present with fever, cough, chest pain, and shortness of breath.
 - c. Cause is multifactorial and includes both infection and infarction of pulmonary tissue.
 - d. **Diagnostic evaluation:** hypoxia and new chest radiograph abnormalities
 - e. **Treatment**
 - (1) Supplemental oxygen, hydration, and antibiotics
 - (2) Exchange transfusion to reduce the proportion of hemoglobin S may also be needed.

4. Strokes
 - a. May be thrombotic or hemorrhagic (intracranial or subarachnoid)
 - b. Clinical presentation: patients usually present with abrupt onset of aphasia, hemiparesis, change in vision, and/or seizures.
 - c. Diagnostic evaluation: baseline laboratory studies and CT or MRI of the brain
 - d. Treatment: immediate exchange transfusion to decrease the amount of hemoglobin S to <30%.
5. Arthropathy: suspect infection, osteomyelitis (especially *Salmonella*) or gout
6. Renal papillary necrosis (gross hematuria and flank pain)
7. Thrombotic or fat emboli
8. Potential sequelae
 - a. CHF (chest radiograph → bibasilar congestion)
 - b. Renal insufficiency/nephrotic syndrome
 - c. Leg ulcers (particularly on the malleoli)
 - d. Retinal infarction/detachment
 - e. Avascular necrosis
 - (1) Digits (children)
 - (2) Femoral head (young adults)

V. ANEMIA

A. General approach

1. Decide how emergent and prioritize decision making.
2. Profound nontraumatic anemia causing severe compromise to the patient's physiologic functions: attention should be focused first on resuscitation, then on diagnosis of underlying causes.

B. Clinical presentation

1. History
 - a. Symptoms of anemia: chest pain, exertional dyspnea, syncope
 - b. Bleeding diathesis
 - (1) Bleeding after trauma, surgery, tooth extractions, or injections
 - (2) Spontaneous bleeding: epistaxis, menorrhagia
 - (3) Spontaneous purpura, petechiae
 - c. Intermittent jaundice/dark urine
 - d. Dietary history: vegetarian diet or poor nutrition
 - e. Drug use/toxin exposure
 - f. Family history and racial background: African-American, Middle Eastern
 - g. Underlying disease
 - (1) Renal or liver disease
 - (2) Hypothyroidism
 - (3) Chronic disease states: cancer, rheumatic or renal disease

2. Physical examination

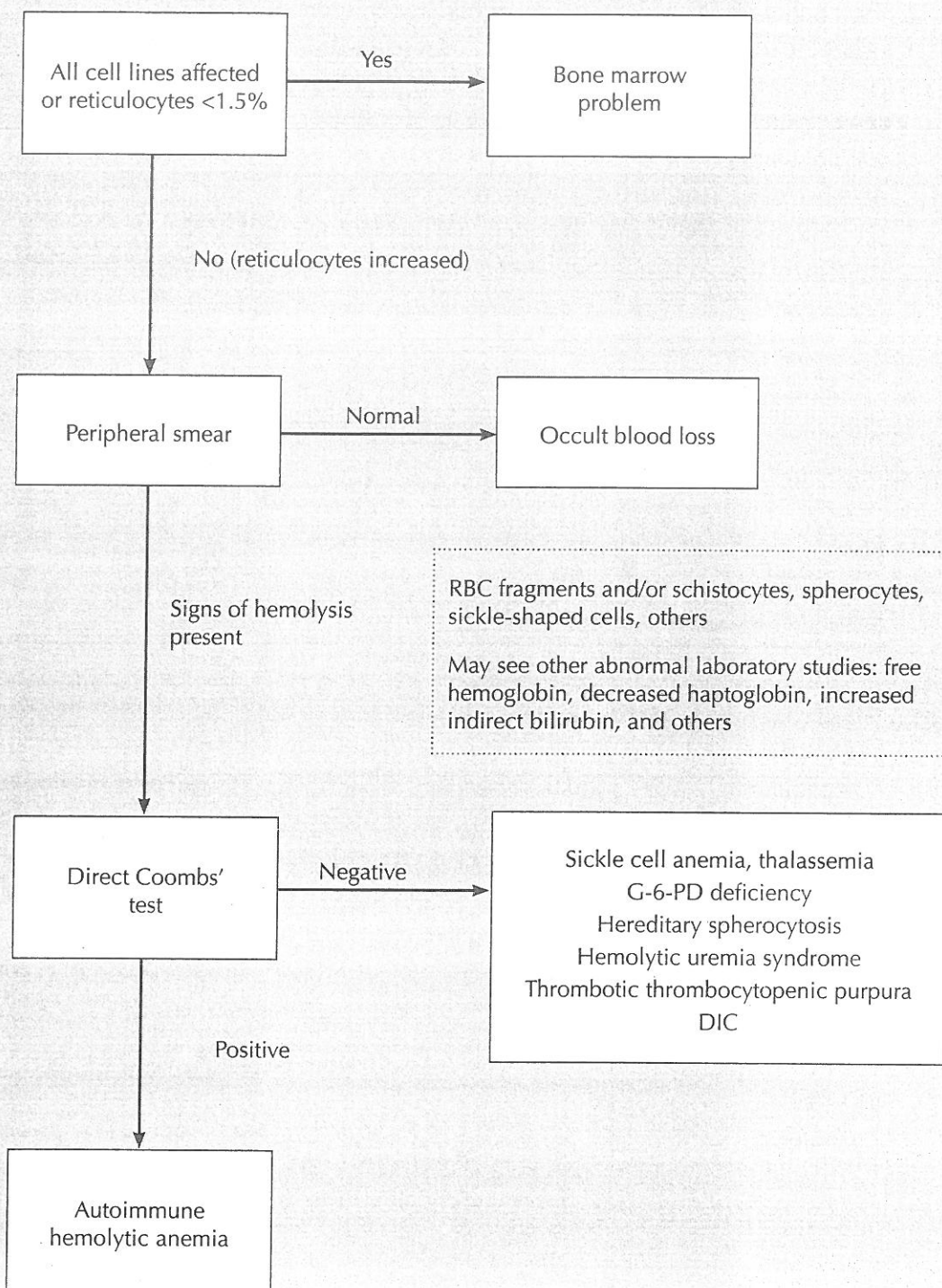
- a. Skin: pallor, purpura, petechiae, angiomas, ulcerations
- b. HEENT
 - (1) Conjunctival jaundice/pallor/hemorrhage, funduscopy hemorrhage
 - (2) Tongue atrophy, papillary soreness
- c. Cardiopulmonary
 - (1) Heart size, murmurs, extra-cardiac sounds
 - (2) Wheezing, rales, other signs of pulmonary edema
- d. Abdomen: hepatomegaly, ascites, splenomegaly, masses, rectal/pelvic
- e. Neurologic: altered position or vibratory sense, peripheral neuritis

C. Diagnostic evaluation

- 1. Basic laboratory studies should be obtained: CBC with differential, platelet count, mean corpuscular volume (MCV), reticulocyte count, peripheral smear
- 2. Other tests can be ordered as indicated.
 - a. Suspect hemolytic anemia: direct Coombs' test, serum free hemoglobin and haptoglobin
 - b. Suspect microangiopathic hemolytic anemia (MAHA) or DIC: PT/PTT, d-dimer, fibrin degradation products, fibrinogen

Table 39: Differential Diagnosis of Anemia

Mechanism/Type	Examples
Blood loss	
Traumatic	
Nontraumatic	GI/GU bleeding, menorrhagia, aneurysmal rupture, hemoptysis
Decreased production	
Hypochromic/microcytic	Iron deficiency, thalassemia, sideroblastic anemia or lead poisoning, chronic disease (cancer, renal, inflammatory diseases)
Macrocytic	B ₁₂ /folate deficiency, liver disease, hypothyroidism
Normocytic	Primary bone marrow involvement: aplastic anemia, myelofibrosis Underlying disease: hypoendocrine state (thyroid, adrenal, pituitary), uremia, liver disease, chronic inflammation
Increased destruction (hemolytic anemias)	
Intrinsic	Enzyme defects: pyruvate kinase deficiency, G6PD deficiency Membrane abnormality: spherocytosis/elliptocytosis, paroxysmal nocturnal hemoglobinuria, spur cell anemia Hemoglobin abnormality: hemoglobinopathies, thalassemias, unstable hemoglobin, hemoglobin M
Extrinsic	Immunologic: allo/autoantibodies Mechanical: microangiopathic hemolytic anemia, prosthetic heart valves Environmental Drugs/toxins Infections Thermal Abnormal sequestration: hypersplenism

**Anemia Present Without Obvious Site of Blood Loss Detected**

Courtesy of Colin Kaide, MD

D. Nonhereditary anemia: hemolytic anemia

1. Acquired anemias
 - a. Precipitating factor is extrinsic to the body.
 - b. Result of exposure to some immunologic, toxic/infectious, or mechanical problem
2. Autoimmune hemolytic anemia
 - a. **The direct Coombs' antiglobulin test is used to demonstrate IgG or complement (C3) attached to the surface of the RBCs: positive only in immune-mediated hemolytic anemias.**
 - b. **Indirect Coombs' test looks for free-circulating antibodies and is used in pretransfusion screening.**
 - c. Warm and cold antibody hemolytic anemia exist.
 - d. **Drug-induced hemolytic anemias**
 - (1) **Many drugs have been linked with induction of hemolytic anemias via various mechanisms.**
 - (2) **The prototype drugs for each reaction are alpha-methyldopa, procainamide, penicillin, and quinidine.**
 - (3) **Hemolysis generally resolves when the drug is stopped.**
3. Fragmentation hemolysis: MAHA
 - a. Thrombotic thrombocytopenic purpura/hemolytic uremic syndrome
 - b. Pregnancy-associated hemolysis
 - (1) Preeclampsia, eclampsia, or placental abruption can result in MAHA.
 - (2) Preeclampsia (even with minimal signs and symptoms) can be associated with the HELLP syndrome, which untreated can lead to liver failure, DIC, or CHF.

Hemolysis
Elevated Liver enzymes
Low Platelets
4. Toxic hemolysis
 - a. Infectious agents
 - (1) Malaria, babesiosis, and bartonellosis can cause hemolysis via parasitization of the RBCs.
 - (2) *Hemophilus influenzae* type B can alter RBC membranes, which leads to their destruction by the immune system.
 - (3) *Clostridium perfringens* releases enzymes that break down RBC membranes.
 - (4) *Mycoplasma* and mononucleosis induce cold agglutinins.
 - b. Other toxins: insect and snake bites (especially cobras) can induce hemolysis; American pit vipers usually cause coagulopathies.
5. Mechanical destruction: heat, repetitive trauma to hands or feet and shearing by passage of blood through oxygenators or artificial heart valves can induce breakage of RBC membranes.

E. Acquired nonhemolytic anemias

1. General
 - a. Problems with production of RBCs because of absent components (iron deficiency) or marrow disease
 - b. Characterized by a decreased reticulocyte count
 - c. The MCV and iron studies can help to further classify and differentiate anemias.

2. Aplastic anemia
 - a. Disease of stem cells in the bone marrow that results in a pancytopenia
 - b. Most cases are idiopathic, but up to 20% are associated with drug or chemical exposure.
 - (1) Benzene
 - (2) Phenylbutazone, gold, D-penicillamine
 - (3) Anticonvulsants
 - (4) Sulfonamides, chloramphenicol
 - (5) Clozapine (antipsychotic)
 - (6) Chemotherapeutic agents
 - c. 10% are due to viral infections, especially hepatitis, Epstein-Barr virus, cytomegalovirus
 - d. Management
 - (1) Best handled by a hematologist; is often supportive but can include bone marrow transplant, immunosuppression, and transfusions
 - (2) Fever in a neutropenic patient (absolute neutrophil count <500 cells/mm³) and severe thrombocytopenia are medical emergencies.
3. Iron-deficiency anemia
 - a. Results from blood loss (often GI or menstrual), increased iron requirements (pregnancy, lactation), or malabsorption of iron
 - b. Typically a microcytic anemia; a normal MCV may be seen early on.
 - c. Iron studies rarely help the emergency physician but are often requested by consultants.
 - (1) Serum ferritin
 - (a) <12 mcg/L indicates low iron stores
 - (b) Also an acute phase reactant; can be increased in inflammatory states
 - (2) Serum iron is low (<60 mcg/dL).
 - (3) Total iron binding capacity is high (>360 mcg/dL).
 - d. Before treating a patient with iron supplements, the cause of iron deficiency should be identified.
4. Megaloblastic anemias
 - a. Macrocytic anemias (increased MCV) all result from problems with DNA synthesis and altered cell morphology.
 - b. Virtually all cases result from folate or vitamin B₁₂ deficiency.
 - c. Patients with inadequate nutrition or malabsorption syndromes rarely require emergency department treatment.
5. Myelodysplastic syndromes
 - a. Acquired clonal disorders of hematopoietic stem cells
 - b. Sideroblastic anemia, characterized by abnormal RBC iron metabolism, can result from toxicity of lead, isoniazid, chloramphenicol, and ethanol.
6. Other anemias
 - a. Anemia of renal disease
 - (1) Decreased endogenous erythropoietin production
 - (2) Treated with exogenous erythropoietin
 - b. Anemia of chronic disease: patients with long-standing inflammatory and autoimmune disease, malignancy, or chronic infections

F. Hereditary anemia

1. General

- a. Normal hemoglobin (Hb A)
 - (1) Four polypeptide chains—2 alpha and 2 non-alpha
 - (2) Variation occurs in the non-alpha chains
- b. In normal individuals
 - (1) 97% of the population has 2 beta chains + the constant 2 alpha chains (a₂ b₂)
 - (2) Some rare normal individuals have gamma or delta chains (a₂ g₂ or a₂ f₂)

2. Thalassemias

- a. In a thalassemia, problems in coding for the hemoglobin chains occur, and variable amounts of the finished product (Hb A) are made.
- b. Microcytic anemias
- c. Common in people of Mediterranean and African heritage
- d. Alpha-thalassemias
 - (1) Severity depends on the number of globin genes that are absent or defective.
 - (2) Can range from mild to fetal death
- e. Beta-thalassemias (Bthal) are classified as major or minor, depending on how much normal Hb A is produced.
 - (1) Bthal minor: chronic anemia of little consequence
 - (2) Bthal major: severe anemia and complications (related to disease)

VI. BLOOD TRANSFUSIONS

A. Transfusion of specific blood products

1. Packed RBCs

- a. Indicated for red cell repletion (not volume repletion)
- b. Transfusion of packed RBCs coupled with crystalloid fluid resuscitation has become the standard of care for the treatment of the acutely hemorrhaging patient.
- c. Type O negative (universal donor) should be given for exsanguinating hemorrhage if type-specific blood is not immediately available, ie, if you ordered it, and you are losing the patient, don't wait for it; as soon as type-specific blood is ready, you can switch infusions if need be.
- d. One unit of packed cells is 250 mL volume; standard practice is to administer at least two units in adults, 15 mL/kg in children.
- e. Packed RBCs can be leukocyte-reduced, washed, irradiated, or frozen for specific needs. Consider leukocyte-reduced packed cells for:
 - (1) Protecting immunocompromised patients
 - (2) Preventing sensitization
 - (3) Preventing nonhemolytic febrile reactions

2. Fresh frozen plasma (FFP)

- a. Contains all the coagulation factors except platelets
- b. Indications

- (1) Correction of clinically significant depletion of clotting factors, eg, patient on warfarin therapy who is actively bleeding or requires emergent surgery
 - (2) Treatment of an acute undiagnosed bleeding disorder
 - c. Requires ABO matching and carries risk of infectious disease transmission
 - d. Typical dosage is 4 units (250 mL/unit) for adults, 15 mL/kg for children.
3. Platelets
- a. Indicated for spontaneous bleeding resulting from thrombocytopenia or inadequate platelet function
 - b. A platelet pack of 6 units (typical adult transfusion) raises the platelet count $\sim 50,000/\text{mm}^3$.
 - c. ABO matching is preferred; platelets may also be leukocyte-reduced.
 - d. Transfusion is associated with risk of infectious disease transmission.
 - e. Contraindications: TTP and HIT
 - f. Ordering platelets made simple
 - (1) Platelets are ordered in packs, not individual units.
 - (2) Some hospitals use 4 packs, and some use 6 packs. This equals either 4 or 6 individual units of platelets, usually from random donors.
 - (3) The 4/6 pack raises the platelet count by $\sim 50,000$.
 - (4) Platelets can also be ordered as an apheresis unit. This is made from one donor, from whom a lot of blood is taken. A certain amount of platelets are removed from the blood, and the rest of the blood and plasma is returned to the donor.
 - (5) An apheresis unit should raise the platelet count by $\sim 50,000$ and expose the patient to fewer antigens and possible transmissible infections.
4. Cryoprecipitate
- a. Contains large quantities of factor VIII:C, vWF, vWF:Ag, and fibrinogen (factor I)
 - b. Effective in bleeding due to:
 - (1) vWD: effective for all types of vWD but is not the treatment of first choice
 - (2) Hemophilia A (not the therapy of first choice)
 - (3) Hypofibrinogenemia (congenital or acquired)
 - c. Does not require ABO matching
 - d. Use is associated with risk of transmission of both hepatitis and AIDS.
5. Factor concentrates/plasma coagulation factors (contain no fibrinogen) are used in management of coagulation factor deficiencies.
- a. Contain high concentrations of the deficient factor and therefore are particularly useful in patients with hemophilia
 - (1) Hemophilia A \rightarrow factor VIII concentrate
 - (2) Hemophilia B \rightarrow factor IX concentrate
 - (3) vWD \rightarrow factor VIII concentrate containing large amounts of vWF
 - b. Almost completely free of the risk of transmission of both the hepatitis and AIDS viruses
6. Albumin and plasma protein fraction are no longer used because of the lack of proven efficacy of volume resuscitation; crystalloid administration is preferable.

B. Transfusion methods

1. Blood products should be infused with warmed normal saline (hypotonic saline solutions; solutions containing calcium or glucose should not be used). Whole blood and packed RBCs should be typed and crossmatched. In certain situations, however, untyped or non-crossmatched blood must be given:
 - a. When type-specific blood is not available in a patient with exsanguinating hemorrhage, type O-negative blood should be given.
 - b. Type-specific blood is indicated for:
 - (1) Progressive hypovolemic shock in spite of aggressive shock management (lactated Ringer's or normal saline 2 L over 10–15 minutes)
 - (2) Profound hypovolemic shock unresponsive to aggressive shock management in the first 10–15 minutes of therapy
2. Autotransfusion
 - a. This is a patient-specific source of blood for the patient in urgent need who has a traumatic hemothorax. (Blood collected from the chest does not have normal levels of fibrinogen nor does it have normally functioning platelets.)
 - b. Technique: blood is drained through a chest tube, screenfiltered to remove clots and debris, then reinfused through a micropore filter.

C. Complications of transfusions

1. Complications associated with massive transfusions: defined as the transfusion of a volume of blood equivalent to the patient's entire blood volume within a 24-hour period or the transfusion of the equivalent of one-half of the patient's blood volume at one time.
 - a. Coagulopathies
 - (1) May result from dilution of clotting factors, platelet destruction, or DIC
 - (2) Administration of FFP should be reserved for the massively transfused and bleeding patient who is in shock. Routine prophylactic use of FFP is unwarranted.
 - (3) Platelets should be administered to patients who receive a massive transfusion in <6 hours and require ongoing transfusion.
 - b. Hypothermia
 - (1) May be caused by transfusion of multiple units of cold blood and is associated with significant morbidity
 - (2) Decreases cardiac output, increases cardiac irritability, and can precipitate ventricular fibrillation. It also shifts the oxygen hemoglobin dissociation curve to the left, thereby decreasing the delivery of oxygen to the cells.
 - (3) Administration of warmed blood can prevent this complication.
 - c. Microembolization
 - (1) Occurs when microaggregates form in refrigerated blood from the degeneration products of platelets, leukocytes, and fibrin
 - (2) Embolization of these aggregates is thought to be associated with development of ARDS.
 - (3) Use of micropore filtration will decrease the incidence of this complication.
 - d. Hypocalcemia (citrate toxicity)
 - (1) Decreased Ca^{++} can occur, because citrate chelates ionized calcium until all the citrate is metabolized.
 - (2) Routine prophylactic calcium supplementation, however, is not recommended.

- (3) Calcium supplementation should be reserved for those patients who develop evidence of myocardial impairment.
2. **Complications associated with any transfusion**
 - a. **Immediate reactions**
 - (1) Hemolytic reaction (most worrisome): an antibody-mediated reaction characterized by the development of fever, chills, burning at the infusion site, joint and low back pain, chest tightness, bleeding, hematuria, and shock
 - (2) Febrile reaction (most common and least worrisome): characterized by fever, chills, and malaise, but patients may occasionally go on to develop blood pressure and respiratory distress
 - (3) Allergic reaction (rare): characterized by development of urticaria skin flushing, laryngeal edema, and/or shock
 - (4) Altered hemoglobin function: storage of blood \rightarrow \downarrow 2,3 diphosphoglycerate levels \rightarrow increased affinity of hemoglobin for oxygen \rightarrow decreased delivery of oxygen to tissues
 - (5) Hyperkalemia and acidosis: may occur in association with massive transfusions
 - b. **Delayed reactions**
 - (1) Infections: many different blood-borne infections may be transmitted by transfusions, including hepatitis, AIDS, human T-cell lymphotropic viruses, cytomegalovirus, Epstein-Barr virus, and malaria. Hepatitis C (formerly non-A, non-B) is currently the most common infection acquired from blood transfusions.
 - (2) Extravascular hemolysis: most commonly manifests as an unexplained decrease in the hemoglobin level 7–10 days after transfusion
 - (3) Graft versus host disease

VII. REVERSAL OF DRUG-INDUCED ANTICOAGULATION

A. Reversal of warfarin-induced coagulopathy

1. **General**
 - a. Increased INRs <9 do not need to be routinely corrected in the absence of bleeding.
 - b. In some special populations, such as patients with artificial heart valves, left ventricular assist devices, or active clots, the benefits of reversing anticoagulation in cases of minor bleeding should be weighed against the possibility of causing more harm by increasing clot burden. With significant trauma or serious hemorrhage, any warfarin effect should be reversed.
 - c. Warfarin is a vitamin K antagonist. It blocks the synthesis of active forms of the vitamin K–dependent coagulation factors II, VII, IX, and X.
2. **Reversal agents**
 - a. Warfarin-induced anticoagulation is reversed by adding back into the system the missing factors (II, VII, IX, and X) and by providing additional vitamin K to allow the liver to produce new factors.
 - b. Vitamin K is given orally or IV. *SC or IM injections are not recommended, because absorption is erratic.* The risk of serious or life-threatening reactions from IV vitamin K is very low. INR correction begins in 2 hours and is usually completed within 24 hours (see Table 40 for dosing recommendations).

- c. FFP is given to replace the missing factors II, VII, IX, and X. The minimum dose should be 4 units. With FFP, the lowest attainable INR is 1.5. This is because the IR of FFP is 1.5.
- d. Prothrombin complex concentrates (PCC) have been used in Europe and other parts of the world for years as reversal agents. PCCs are highly purified concentrates of the vitamin K-dependent clotting factors II (prothrombin), VII, IX, and X. They are pooled from plasma and although purified, do carry a very small risk of viral transmission. Product formulations vary in concentrations of each factor. They can be 3-factor preparations with minimal VII activity or 4-factor with good VII levels. In the United States, two PCC products are available: Profilnine SD® and Bebulin VH®. They both have very low factor VII activity and are 3-factor preparations. European preparations usually are 4-factor preps. Some also contain protein C and S to limit the risk of inducing inappropriate clotting.
 - (1) All factors are referenced against 100 U of factor IX activity.
 - (2) All preparations are dosed based on the amount of factor IX.
 - (3) There are reports of thrombosis with PCC preparations; however, the risk appears low.
 - (4) Onset is very fast (15 minutes), and INRs can return to normal in 10 minutes.
- e. Factor eight inhibitor bypassing activity (FEIBA)
 - (1) FEIBA is used in patients with hemophilia who develop antibodies to factor VIII after receiving factor VIII replacement for a prolonged time.
 - (2) It is an activated form of PCC (activated II, VII, IX, and X).
 - (3) It has been shown to be an effective reversal agent for warfarin-induced anticoagulation. It may have a thromboembolic risk greater than that of PCC.
 - (4) There is no FDA-approved indication for FEIBA as a warfarin reversal agent.
- f. Activated factor VII
 - (1) A very large thrombin burst is theoretically possible to create by directly activating factor X using recombinant factor VII (rVIIa).
 - (2) The INR can be rapidly normalized, although redosing is needed every few hours.
 - (3) Despite normalizing the INR, some studies have called into question the effects of rVIIa on actual clinical reversal of bleeding.
 - (4) There is no FDA-approved indication of rVIIa as a warfarin reversal agent.

Table 40: Recommendations for Managing Increased INRs or Bleeding in Patients Receiving Vitamin K Antagonists

Condition	Description
INR above therapeutic range but <5.0; no significant bleeding	Lower dose or omit dose, monitor more frequently, and resume at lower dose when INR therapeutic; if only minimally above therapeutic range, no dose reduction may be required.
INR \geq 5.0 but <9.0; no significant bleeding	Omit next one or two doses, monitor more frequently, and resume at lower dose when INR in therapeutic range. Alternatively, omit dose and give vitamin K ₁ (\leq 5 mg orally), particularly if at increased risk of bleeding. If more rapid reversal is required because the patient requires urgent surgery, vitamin K ₁ (2–4 mg orally) can be given with the expectation that the INR will decrease in 24 hours. If the INR is still high, additional vitamin K ₁ (1–2 mg orally) can be given.
INR \geq 9.0; no significant bleeding	Hold warfarin therapy and give higher dose of vitamin K ₁ (5–10 mg orally) with the expectation that the INR will be reduced substantially in 24–48 hours. Monitor more frequently, and use additional vitamin K ₁ if necessary. Resume therapy at lower dose when INR therapeutic.
Serious bleeding at any elevation of INR	Hold warfarin therapy and give vitamin K ₁ (10 mg by slow IV infusion), supplemented with fresh plasma or prothrombin complex concentrate, depending on the urgency of the situation; recombinant factor VIIa may be considered as alternative to prothrombin complex concentrate. Vitamin K ₁ can be repeated every 12 hours.
Life-threatening bleeding	Hold warfarin therapy and give prothrombin complex concentrate, supplemented with vitamin K ₁ (10 mg by slow IV infusion); recombinant factor VIIa may be considered as alternative to prothrombin complex concentrate. Repeat if necessary, depending on INR.

Note: If continuing warfarin therapy is indicated after high doses of vitamin K₁, then heparin or LMWH can be given until the effects of vitamin K₁ have been reversed and the patient becomes responsive to warfarin therapy. It should be noted that INR values >4.5 are less reliable than values in or near the therapeutic range. Thus, these guidelines represent an approximate guide for high INRs.

Source: Ansell J, Hirsh J, Hylek E, et al. Pharmacology and management of the vitamin K antagonists: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (8th Ed). *Chest*. 2008;(6 Suppl):175S. Used with permission from the American College of Chest Physicians.

B. Reversal of heparin and low-molecular-weight heparin (LMWH)

1. General

- Heparin joins with antithrombin III (ATIII), and the complex binds both factor II and factor X in a 1:1 ratio, blocking two critical parts of the clotting cascade.
- LMWH, a piece of the heparin molecule, joins with ATIII, and the complex binds both factor II and factor X in a 1:3 to as high as a 1:5 ratio. Owing to the high binding of factor X, an "anti-Xa" level must be used if the effect of LMWH on the clotting system needs to be measured (rarely if ever needed in the emergency department). PTT does not accurately predict the degree of anticoagulation with an LMWH. Currently available LMWHs include enoxaparin, dalteparin, and tinzaparin.
- Another synthetic polysaccharide that is often, but not correctly, categorized as an LMWH is fondaparinux. It is a Xa-only inhibitor that also works via ATIII.

2. Reversal agent (protamine)

- a. Protamine sulfate was derived from the sperm of fish (salmon) and is now recombinant. The mechanism of action is to bind to and inactivate heparin. The heparin-protamine complex is removed by the reticuloendothelial system. Protamine alone has a weak anticoagulant effect by inhibiting factor V.
- b. Doses should not exceed 50 mg at a time.
- c. Protamine may have some effect on LMWH. Only 60%–75% of the anti-Xa activity of LMWH is neutralized by protamine. Effectiveness depends on which LMWH is used.
- d. There is a real concern when using protamine with LMWH—protamine when given by itself has anticoagulant effects. If there is reversal of the non-Xa activity and only partial (but not enough) reversal of the Xa activity, the net result will point to anticoagulation.

C. Reversal of newer anticoagulants

1. Data supporting the reversal of the newer anticoagulants rivaroxaban and dabigatran is limited to ex vivo and in vitro studies. There are as of yet no randomized, controlled trials supporting the reversal of these agents.
2. Rivaroxaban (and soon apixaban) are direct factor Xa inhibitors, ie, they bind directly to factor Xa without using ATIII.
3. Dabigatran is a direct factor II inhibitor, ie, it binds directly to factor II without using ATIII.
4. These agents attack at the most critical part of the clotting cascade, making reversal difficult.
5. Information on reversal with the following agents is not FDA approved, but it does appear on many written protocols. It represents the best information available at the time of this publication, until accepted protocols become standard.
6. Strategies that have been tried involve overwhelming the factor inhibition by introducing a large amount of replacement factors into the system. PCC and aPCC (FEIBA) have been tried for both agents. FEIBA thus far has shown promise in reversal of both agents. These studies have been conducted in volunteers and in animals and are not definitive.
7. In theory, rVIIa may also work to reverse the effect of factor X inhibition by direct inhibitors. The possible reversal of rivaroxaban by rVIIa has been suggested because of its ability to reverse the indirect Xa inhibitor fondaparinux.

D. Reversal of thrombolysis

1. General
 - a. Thrombolytics (rTPA and others) work by catalyzing the conversion of plasminogen to plasmin. Plasmin cleaves cross-linked fibrin into d-dimers and fibrin degradation products.
 - b. Reversal is not an accurate term in that the best that can be done is to mitigate the effects as best as possible.
2. Agents to "reverse" thrombolytic-induced bleeding
 - a. ϵ -Aminocaproic acid
 - (1) Blocks fibrinolysis by reversibly blocking plasminogen, which then cannot be cleaved to plasmin; plasmin cleaves crosslinked fibrin and lyses clots.
 - (2) Enhances hemostasis when fibrinolysis contributes to bleeding; used for bleeding in patients with hemophilia after dental procedures
 - (3) There are no data on the use for rescue of rTPA (or other thrombolytics) for bleeding in the brain or other sites when rTPA is used for stroke, MI, or pulmonary embolism.
 - (4) Not FDA approved for this use

b. Cryoprecipitate

- (1) Contains a significant amount of fibrinogen (see above for details)
- (2) Gives back the fibrinogen that is degraded by the action of tPA

Table 41: Anticoagulant Reversal Agents

Anticoagulant	Mechanism of Anticoagulation	Reversal Agents
Warfarin	Interferes with the production of II, VII, IX, X	Vitamin K, FFP, PCC, aPCC
Heparin	Potentiates the action of antithrombin, inactivating Xa and thrombin	Protamine
LMWH	Acts via antithrombin to inactivate Xa and thrombin. Activity varies but mostly anti-Xa activity	Protamine (partial), rVIIa
Synthetic pentasaccharide (fondaparinux)	Indirectly inhibits Xa via antithrombin	rVIIa, PCC, aPCC may be effective but limited data
Direct thrombin inhibitors	Inhibit thrombin without using antithrombin	rVIIa, PCC, aPCC may be effective but limited data
Direct Xa inhibitors	Inhibit Xa without using antithrombin	
Platelet inhibitors (eg, clopidogrel)	Block various mechanisms by which platelets aggregate and degranulate	Platelets (?), DDAVP (?), rVIIa (?)
Thrombolytics	Catalyze plasminogen to plasmin, causing clot lysis	Aminocaproic acid, cryoprecipitate

HEMATOLOGIC DISORDERS: PRACTICE CLINICAL SCENARIOS

Answers immediately follow the practice clinical scenarios.

Scenario A

Presentation: A patient presents to the emergency department with altered mental status. He is confused and lethargic.

Laboratory studies: Platelet count is 75,000/mm³, and hemoglobin is 9.5 mg/dL. There has been no heparin exposure. The laboratory reports schistocytes on the peripheral blood smear.

What is the diagnosis?

Scenario B

Presentation: A patient with sickle cell disease presents with complaints of chest pain. He complains of discomfort with breathing, shortness of breath, and fever.

What is the diagnosis?

Scenario C

Presentation: A patient presents 3 days after being discharged from the hospital after a left knee replacement. The patient is on a LMWH 40 mg/day (prophylactic dosage).

Physical examination: The patient has what appears to be bleeding at the sites of the LMWH injections. She also has a painful, pulseless lower extremity on the right side (the opposite side from the knee replacement).

What is the diagnosis?

Scenario D

Presentation: A patient with hemophilia A comes in with swelling and pain in his knee after a minor injury. He took some of his factor at home but still complains of increased swelling and worsening pain.

What is the diagnosis?

Scenario E

Presentation: A patient is on warfarin and was started on TMP-SMX. She presents with a massive GI bleed.

Laboratory studies: The patient's INR is 9. Her INR before starting an antibiotic was 2.4. Her hemoglobin B is 9 mg/dL.

What is the diagnosis?

ANSWERS TO PRACTICE CLINICAL SCENARIOS

Scenario A

Diagnosis: thrombotic thrombocytopenic purpura (TTP)

Diagnostic evaluation: Never ignore schistocytes on a peripheral smear; they are always bad! If $>1\%$ is reported, consider a microangiopathic hemolytic anemia. This patient meets the three most important criteria for TTP: thrombocytopenia, neurologic symptoms (due to microthrombi), and schistocytes on the peripheral blood smear (due to breakage of cells flowing through platelet plug debris). Fever and renal abnormalities are less common. Two-thirds of patients with TTP have some neurologic symptoms due to microthrombi in the CNS.

Management: The main treatment is plasma exchange transfusion, which is done by a hematologist. Additional management includes steroids to decrease production of the pathologic antibody (against ADAMTS13) and FFP to replete ADAMTS13.

Scenario B

Diagnosis: sickle cell with chest pain and shortness of breath

Diagnostic evaluation: The patient's oxygen saturation is 90%. Chest radiograph shows a patchy lower lobe infiltrate on the right side. Although this could be nothing more than a community acquired pneumonia, you must be concerned about acute chest syndrome, which is seen in SS, SC, sickle-thalassemia. The criteria for diagnosis are new pulmonary infiltrate involving at least one complete lung segment consistent with alveolar consolidation but excluding atelectasis, *plus at least one or more clinical findings of cough, wheezing, tachypnea, temperature $>38.5^{\circ}\text{C}$ (101.3°F), dyspnea/hypoxia, or chest pain.*

Management: Testing should include a radiograph to look for infiltrate. Treatment is aimed at treating infection and maximizing symptom control and respiratory function. This can include bronchodilators, transfusions, pain medications, and respiratory support as needed.

Scenario C

Diagnosis: heparin-induced thrombocytopenia

Diagnostic evaluation: Although the patient has a platelet count that is seemingly normal ($130,000/\text{mm}^3$), postoperative platelet counts should be increased. Her platelet count on the last known laboratory study is $600,000/\text{mm}^3$, which would be normal in the postoperative state. She has had a drop of $>50\%$. Remember the T's: The patient has thrombocytopenia (relative), thrombosis, and no other explanation. The timing is within the appropriate range of 5–10 days.

Management: Stop the LMWH (or regular heparin). An alternative form of anticoagulation is needed. Because the patient will be admitted, start a drug like argatroban. If the patient had been on warfarin, vitamin K therapy would be needed. There is no need for fresh frozen plasma.

Scenario D

Diagnosis: hemophilia

Key facts:

- Bleeding history in a hemophiliac
- Bleeding after mild trauma or dental extractions
- Spontaneous hematuria
- Hemarthrosis and muscle hematomas (90% of all bleeding)
- Intracranial hemorrhages (a major cause of death in patients with hemophilia A)

Management: Treatment includes replacement of the missing factor. Factor VIII concentrate is the treatment of choice. Check the patient's record or ask the patient how much factor he would normally get in the situation. Patients with hemophilia very likely know a lot more about it than you do, because they have lived with it their entire life, whereas you may not see it very often. Dosing is determined by whether the bleeding is mild, moderate, or severe.

- In mild bleeding (hematuria, early hemarthrosis, deep laceration), the goal is to increase factor levels by 25% → 12.5 units/kg.
- In moderate bleeding (oral lacerations, dental extraction or minor surgery, late hemarthrosis), the goal is to increase factor levels up to 50%. → 25 units/kg.
- In severe bleeding (CNS, GI, intra-abdominal or retroperitoneal bleeding; major trauma or surgery; documented or potentially serious head injury), the goal is to increase factor levels up to 100% → 50 units/kg.

Scenario E

Diagnosis: warfarin over-anticoagulation

Alternative presentation: A patient on warfarin is sent over from his family doctor's office for an asymptomatic INR of 6.

Diagnostic evaluation: Determine the need for reversal of anticoagulation. Severe bleeding with an increased INR almost always needs to be treated. Caution should be exercised in patients with a left ventricular assist device or mechanical artificial valve; these are not absolute contraindications to reversal, but consider discussing with the cardiovascular surgeon to establish a target final INR. In cases when the acute bleeding can be controlled (cauterized epistaxis, banded varices, etc), reversal of warfarin should be followed by cross-anticoagulation with heparin. Patients with increased INRs who are asymptomatic may be treated by holding a dose of warfarin ± small doses of vitamin K.

Management: If needed, administer vitamin K orally or IV. Replace missing factors when indicated. Fresh frozen plasma or PCC can be used.

ONCOLOGIC DISORDERS

Upper Airway Obstruction.....	947
Tamponade Secondary to Malignant Pericardial Effusion.....	947
Superior Vena Cava Obstruction	948
Acute Spinal Cord Compression.....	949
Hypercalcemia Secondary to Malignancy	950
Syndrome of Inappropriate ADH Secretion (SIADH)	951
Acute Tumor Lysis Syndrome.....	952
Hyperviscosity Syndrome.....	953
Adrenal Insufficiency	954
Granulocytopenia, Immunosuppression, and Overwhelming Infection in the Presence of Malignancy	955
Leptomeningeal Carcinomatosis	956

ONCOLOGIC DISORDERS: SELF-ASSESSMENT QUESTIONS

1. All of the following statements regarding cardiac tamponade secondary to malignant pericardial effusion are true except:
 - (a) ECG findings of low QRS voltage and electrical alternans are consistent with this diagnosis.
 - (b) Suggestive physical findings include hypotension with a narrow pulse pressure, jugular venous distention, and muffled heart tones.
 - (c) Supportive measures include fluid restriction and pressors.
 - (d) The diagnosis may be confirmed by echocardiography or Swan-Ganz catheterization.
2. All of the following statements regarding superior vena cava obstruction are accurate except:
 - (a) Patients present with headache, swelling of the face and upper extremities, and shortness of breath.
 - (b) Common causes of this syndrome are lung cancer and lymphoma.
 - (c) Useful therapeutic measures include administration of oxygen, diuretics, and corticosteroids.
 - (d) Initially, symptoms occur in the evening.
3. The ECG finding most consistent with a diagnosis of hypercalcemia is:
 - (a) Shortened QT interval
 - (b) Prolonged PR interval
 - (c) Prolonged QT interval
 - (d) Hyperacute T waves
4. Which of the following signs and symptoms is/are not consistent with the diagnosis of hypercalcemia?
 - (a) Lethargy/decreased level of consciousness
 - (b) Hypotension
 - (c) Constipation
 - (d) Anorexia/nausea/vomiting
5. In a patient presenting with the triad of unexplained stupor or coma, anemia, and rouleaux formation on peripheral blood smear, the most likely diagnosis is:
 - (a) Acute adrenal insufficiency
 - (b) Hypercalcemia
 - (c) Hyperviscosity syndrome
 - (d) Superior vena cava syndrome

6. Which of the following findings is inconsistent with the diagnosis of adrenal insufficiency?
- (a) Hyperkalemia
 - (b) Hypotension
 - (c) Hyponatremia
 - (d) Hyperglycemia
7. The most common presenting symptom in patients with leptomeningeal carcinomatosis is:
- (a) Headache
 - (b) Incontinence
 - (c) Back pain
 - (d) Facial nerve palsy
8. Common causes of the hyperviscosity syndrome include all of the following except:
- (a) Chronic myelocytic leukemia
 - (b) Lymphomas
 - (c) Multiple myeloma
 - (d) Waldenstrom macroglobulinemia
9. The most appropriate initial therapy for malignancy-induced hypercalcemia consists of:
- (a) Administration of glucocorticoids
 - (b) Administration of plicamycin (mithramycin)
 - (c) Fluid hydration with IV normal saline followed by administration of furosemide
 - (d) Administration of calcitonin
10. Infection in the cancer patient is most commonly _____ in etiology.
- (a) Viral
 - (b) Bacterial
 - (c) Fungal
 - (d) Protozoal
11. A reasonable antibiotic regimen for the cancer patient who presents with a history of fever and abdominal pain associated with peritonitis on physical examination is:
- (a) An antipseudomonal aminoglycoside plus an antipseudomonal penicillin
 - (b) A fluoroquinolone and an antipseudomonal aminoglycoside plus an antipseudomonal third- or fourth-generation cephalosporin
 - (c) Metronidazole or clindamycin
 - (d) A combination of A + C or B + C

ANSWERS

- | | | | |
|------|------|------|-------|
| 1. c | 4. b | 7. a | 10. b |
| 2. d | 5. c | 8. b | 11. d |
| 3. a | 6. d | 9. c | |

Use the pre-chapter multiple choice question worksheet (page xx) to record and determine the percentage of correct answers for this chapter.

I. UPPER AIRWAY OBSTRUCTION (AT THE LEVEL OF THE MAINSTEM BRONCHI OR ABOVE)

A. Signs and symptoms

1. Voice change or hoarseness (indicates a slow process of local tumor compression or obstruction)
2. Laryngeal stridor from a rapidly growing tumor
3. Acute upper airway obstruction secondary to infection, tumor hemorrhage, or inspissated secretions; respiratory arrest is imminent.

B. Etiology

1. Carcinoma
 - a. Laryngeal
 - b. Thyroid
 - c. Pulmonary metastases
2. Lymphoma (especially Burkitt)

C. Diagnostic evaluation

1. Immediate assessment
2. Palpation of mass
3. Lateral soft-tissue radiograph of the neck
4. Direct laryngoscopy
5. Fiberoptic laryngoscopy
6. CT if stable

D. Management

1. Establish an airway if there is airway compromise.
 - a. Fiberoptic laryngoscope-assisted intubation can be very useful in securing the difficult airway.
 - b. A surgical airway may be necessary.
2. ENT consult
3. Treat the underlying neoplasm.

II. TAMPONADE SECONDARY TO MALIGNANT PERICARDIAL EFFUSION

A. Clinical presentation

1. Related to the volume of fluid, its rate of accumulation, and the tempo of cardiac compressions
2. Hypotension with narrow pulse pressure
3. Shortness of breath and extreme anxiety and apprehension
4. Jugular venous distention

5. Pulsus paradoxus >10 mmHg (may be absent)
6. Other signs that may be present
 - a. Auscultation: diminished heart sounds
 - b. ECG: low QRS voltage and electrical alternans
 - c. Chest radiograph: enlargement of the cardiac silhouette in association with clear lung fields and a normal vascular pattern (no CHF)

B. Etiology

1. Most common
 - a. Carcinoma of the lung and breast
 - b. Hodgkin and non-Hodgkin lymphoma
 - c. Leukemia
 - d. Malignant melanoma
2. Others
 - a. Mesotheliomas and sarcomas of the pericardium and other metastatic tumors
 - b. Postradiation pericarditis

C. Differential diagnosis

1. Massive pulmonary embolus
2. Acute superior vena cava obstruction

D. Diagnostic evaluation

1. Echocardiography is the diagnostic study of choice; it is sensitive, specific, and noninvasive.
2. Swan Ganz catheterization is performed if diagnostic confirmation is required; a positive finding is diastolic equalization of pressures (pulmonary artery = right ventricle = right atrial = intrapericardial)

E. Management

1. Supportive measures include IV fluids, inotropic support with dobutamine or isoproterenol, and supplemental oxygen
2. Definitive therapeutic measures
 - a. Pericardiocentesis (with ultrasound guidance if possible)
 - b. Pericardial stripping/window surgery
 - c. Radiotherapy/chemotherapy

III. SUPERIOR VENA CAVA OBSTRUCTION

A. Clinical presentation

1. Reflect slow, progressive tumor development; initially, the symptoms occur in early morning.
2. Most common complaints are edema and venous distention of the face and upper extremities plus shortness of breath.
3. Headache or feeling of fullness/congestion in head
4. Facial plethora and telangiectasia
5. Papilledema (signifies a critical increase of intracranial pressure)

B. Etiology

1. Carcinoma of the lung (small-cell or squamous cell): bronchogenic carcinoma is the most common cause of superior vena cava obstruction (usually on the right side).
2. Lymphoma

C. Diagnostic evaluation

1. Chest radiograph: wide mediastinum and/or mass
2. Contrast-enhanced chest CT confirms the diagnosis.
3. MRI is indicated if neck pain is present to exclude coexistent spinal cord compression (Rubin syndrome).
4. Immediate consultation for tissue diagnosis, followed by radiation and/or chemotherapy

D. Management

1. Administer supplemental oxygen.
2. Begin measures to decrease cerebral edema if symptoms are present.
 - a. Elevate head to 30° midline position.
 - b. Diuretics (eg, furosemide)
 - c. Corticosteroids (eg, methylprednisolone)

IV. ACUTE SPINAL CORD COMPRESSION

A. Clinical presentation

1. Back pain (most commonly thoracic) is the initial symptom in 95% of these patients.
2. Pain occurs at the site of tumor metastases; it may be localized or radicular in nature and is usually worsened by percussion over the affected vertebral bodies.
3. Other symptoms include lower extremity weakness, difficulty with ambulation, sensory deficits, paralysis, and urinary incontinence (acute urinary retention may also occur).

B. Etiology

1. Multiple myeloma
2. Lymphoma (Hodgkin or non-Hodgkin)
3. Carcinoma
 - a. Lung (most common cause)
 - b. Breast
 - c. Prostate

C. Diagnostic evaluation

1. Plain radiographs: identify the level of vertebral collapse
2. MRI of spine (diagnostic modality of choice)
3. Myelography
 - a. Associated with significant morbidity resulting from lumbar puncture and dye insertion
 - b. Should be done if MRI is unavailable or if the MR images were of poor quality
4. CT of the spine, in conjunction with myelography, enhances detection of small areas of spinal destruction.

D. Management

1. Steroids (consider high-dose dexamethasone (100 mg); otherwise 4–6 mg every 6 hours) → ↓ inflammation and edema
2. Radiation (the definitive therapy) and surgery (decompression laminectomy) improve neurologic function.

V. HYPERCALCEMIA SECONDARY TO MALIGNANCY (MOST COMMON LIFE-THREATENING METABOLIC DISORDER ASSOCIATED WITH CANCER)

A. Clinical presentation

1. Anorexia, nausea, vomiting
2. Fatigue, weakness
3. Decreased level of consciousness
4. Constipation
5. Hypertension
6. Back pain
7. Polydipsia, polyuria
8. Remember the symptoms as:

Stones (kidney stones)

Bones (osteitis fibrosa cystica: results in pain and pathologic fractures; also can see osteoporosis, osteomalacia, and arthritis)

Groans (abdominal groans: GI symptoms of constipation, indigestion, nausea, and vomiting; hypercalcemia can lead to peptic ulcers and acute pancreatitis)

Moans (psychic moans: lethargy, fatigue, depression, memory loss, psychosis, ataxia, delirium, and coma)

B. Etiology

1. Bone destruction
 - a. Carcinomas of lung, breast, and prostate
 - b. Multiple myeloma
2. Parathyroid hormone-like substance (squamous cell carcinoma of the lung)
3. Osteoclast-activating factor (calcitriol overproduction)
 - a. Non-Hodgkin lymphoma
 - b. Adult T-cell lymphoma or leukemia
4. New hormonal therapy in patients with breast cancer (that is not related to the underlying malignancy)

C. Diagnostic evaluation

1. Serum levels of the following:
 - a. Calcium (↑): check ionized Ca^{++} levels
 - b. Phosphorus (normal or ↓)

- c. Alkaline phosphatase (normal or ↑)
- d. Chloride (<100 mEq/L)
- e. Potassium (↓ in 50% of patients)
- f. BUN/creatinine (↑)
- g. Albumin (↓)
- h. pH changes inversely affect Ca^{++} levels
- 2. ECG: shortened QT interval

D. Management: aimed at decreasing ionized calcium level and preventing bone resorption

- 1. IV normal saline 250–500 mL/hr: clinical improvement often occurs with only fluids and furosemide
- 2. Furosemide 40–80 mg IV every 2–4 hours: used less, commonly in volume-sensitive patients
- 3. Potassium and magnesium replacement as needed
- 4. Bisphosphonates have become the treatment of choice for managing cancer-induced hypercalcemia, supplanting all other pharmacologic approaches except corticosteroids.
 - a. Pamidronate: inhibits osteoclast activity and bone resorption
 - b. Zoledronic acid
- 5. Hydrocortisone 25–200 mg IV every 6–8 hours: useful for certain malignancies (breast cancer, multiple myeloma, lymphoma) but should not be started without oncologic consult, because it decreases intestinal absorption of calcium and vitamin D.
- 6. Other modalities (not usually part of initial emergency department management)
 - a. Calcitonin: particularly useful in patients with severe hypercalcemia associated with an altered mental status and/or renal failure; it decreases the skeletal release of calcium; acts quickly but only small reduction in calcium.
 - b. Hemodialysis: indicated in the presence of profoundly altered mental status, renal failure, or inability to tolerate saline load
 - c. Treatment of underlying malignancy, cessation of hormonal therapy

VI. SYNDROME OF INAPPROPRIATE ADH SECRETION (SIADH)

A. Classic clinical scenario

- 1. Volume expansion without edema (euvolemic hyponatremia)
- 2. Hyponatremia
- 3. Decreased serum osmolality
- 4. Urinary sodium >40 mEq/L and urine osmolality >100–150 mOsm/L associated with a urine specific gravity >1.002
- 5. Normal renal, adrenal, and thyroid functions

B. Clinical presentation

- 1. Depends more on the rate of decrease in serum sodium than on the absolute serum sodium concentration

2. Weakness, malaise
 3. Headache, dizziness
 4. Subtle changes in mental status
 5. Coma, seizures
- C. Etiology: malignancies (medications for malignancies, eg, cyclophosphamide, vincristine, can also cause SIADH)
1. Lung: most common (small cell)
 2. Brain
- D. Diagnostic evaluation
1. Blood
 - a. Electrolytes
 - b. Serum osmolality
 - c. Thyroid function studies
 - d. Adrenal function studies (if myxedema or Addison disease is suspected)
 2. Urine
 - a. Urinalysis
 - b. Sodium
 - c. Creatinine (\uparrow in adrenal disease)
 - d. Osmolality
- E. Management
1. Water restriction
 2. Furosemide 0.5–1 mg/kg with concomitant normal saline results in net free water clearance while maintaining euvolemia.
 3. 3% saline (for seizures/dysrhythmias) 100–250 mL over several hours to lower the risk of central demyelination: 0.5 mEq/L increase in sodium \rightarrow 10–12 mEq in 12–24 hours
 4. Treat underlying tumor.
 5. Demeclocycline: longer-term benefit

VII. ACUTE TUMOR LYSIS SYNDROME

A. Classic clinical scenario

1. Most commonly occurs 1–5 days after chemotherapy for a hematologic malignancy such as leukemia and lymphoma (particularly Burkitt lymphoma) but can happen in any malignancy. Other factors that make acute tumor lysis syndrome more likely include small-cell lung cancer, a high tumor burden, and highly chemosensitive tumors. Biochemical hallmarks include hyperuricemia (DNA breakdown), hyperkalemia (cytosol breakdown), and hyperphosphatemia (protein breakdown). Hypocalcemia is secondary to hyperphosphatemia.
2. The integrity of renal function is a critical factor, because the kidney provides the primary mechanism for excretion of uric acid, potassium, and phosphate.

- a. Acute tumor lysis syndrome is more likely to be seen in patients with preexisting renal insufficiency, and the subsequent metabolic derangements are more likely to be severe than in those with normal renal function.
- b. Acute renal failure may also be precipitated by hyperuricemia and hyperphosphatemia (even in patients with preexisting normal renal function, especially in the presence of rapid tumor lysis).
- c. Hyperuricemia with resultant urate nephropathy is the most common metabolic cause of renal insufficiency.

B. Complications: reflect the type and severity of metabolic derangement

1. Acute renal failure (\uparrow uric acid and/or \uparrow PO_4)
2. Ventricular dysrhythmias, sudden death (\uparrow K^+ or \downarrow Ca^{++})
3. Neuromuscular instability (\downarrow Ca^{++})

C. Management

1. Control preexisting hyperuricemia \rightarrow hydration, allopurinol
 - a. The primary means of reducing hyperuricemia is hydration and diuresis to maintain adequate urinary flow (at least 100 mL/hr).
 - b. Alkalinization is no longer routinely recommended.
2. Rasburicase is now widely used to treat tumor lysis syndrome. It is a recombinant version of a urate oxidase enzyme that is found in many mammals but not people. It catalyzes the conversion of uric acid to allantoin. Discuss with oncologist first, but an empiric dose should be considered in a patient with hyperuricemia and renal insufficiency.
3. Hemodialysis should be considered a potentially life-saving measure.
4. Frequent monitoring of electrolytes, calcium, and phosphorus.

VIII. HYPERVISCOSITY SYNDROME

- A. The degree of viscosity reflects the flow-resisting characteristics of bodily fluids. This syndrome is characterized by marked increase in serum proteins, WBCs, or RBCs, which result in sludging and reduction in microcirculatory perfusion.

B. Classic clinical scenario

1. The patient presents with headache, fatigue, and somnolence or coma. Persistent bleeding from mucosal surfaces is common (and the platelet count is normal). Focal signs of microthrombosis may be present and include deafness, blindness, and seizures.
2. On funduscopic examination, "sausagelinked" retinal vessels, hemorrhages, and exudates may be seen.
3. A clinical clue to this diagnosis is the patient with unexplained stupor or coma who is anemic and demonstrates "rouleaux cells" on the peripheral smear. The laboratory may tell you the chemical tests cannot be performed because of serum stasis in the analyzers, ie, the blood is too thick.

C. Etiology

1. Dysproteinemias
 - a. Waldenström macroglobulinemia

- (1) Most common cause of hyperviscosity syndrome
 - (2) Produces high-viscosity IgM
 - b. Multiple myeloma (IgA and IgG myelomas): second most common cause
 2. Leukemias with blastic transformation, eg, chronic myelogenous leukemia
 3. Polycythemia vera, essential thrombocytosis
- D. Diagnostic evaluation
1. Increased serum viscosity (>4–5 times the viscosity of water)
 2. Increased serum proteins on electrophoresis
 3. WBC count >100,000/mm³ in chronic myelogenous leukemia
 4. Urinalysis → Bence Jones protein in multiple myeloma
- E. Management
1. Dysproteinemias
 - a. Immediate rehydration followed by emergency plasmapheresis
 - b. Phlebotomy of two units and replacement of the patient's RBCs with physiologic saline is appropriate for patients in coma due to hyperviscosity syndrome.
 - c. Chemotherapy
 2. Leukemias with blastic transformation
 - a. Immediate rehydration followed by leukapheresis
 - b. Hydrea
 - c. Chemotherapy

IX. ADRENAL INSUFFICIENCY

- A. Classic clinical scenario
1. Sudden vasomotor collapse is classic.
 2. On physical examination, the patient is hypotensive with signs of dehydration.
 3. Laboratory findings are hypoglycemia, hyponatremia, hyperkalemia, and increased eosinophils.
- B. Etiology
1. Carcinoma of the lung or breast
 2. Withdrawal of chronic steroid therapy
 3. Malignant melanoma
 4. Retroperitoneal malignancies
- C. Management (draw blood for a serum cortisol level first)
1. Aggressive IV fluid therapy with D5 normal saline (1 L over the first hour)
 2. Hydrocortisone 100 mg IV initially and every 6 hours; could use dexamethasone as an alternative if planning ACTH stimulation test.
 3. Correct precipitating factors (especially infection).

X. GRANULOCYTOPENIA, IMMUNOSUPPRESSION, AND OVERWHELMING INFECTION IN THE PRESENCE OF MALIGNANCY

A. Significance

1. Infection is the most common cause of death in the cancer patient and may be subtle in presentation.
2. Fever is usually the initial clinical manifestation of the infectious process and may be the only sign present.
3. The risk of infection increases as the number of granulocytes decreases and is particularly pronounced in the granulocytopenic patient (PMN count $<500/\text{mm}^3$).
4. Thus, fever in the cancer patient should prompt a thorough clinical evaluation and be treated with empiric broad-spectrum antibiotics.

B. Factors that increase the vulnerability of the cancer patient to infection

1. Granulocytopenia
2. Impaired humoral and cellular immunity
3. Chemotherapy-induced immunosuppression and myelosuppression (bleomycin is the most common cause of chemotherapy-induced lung disease)
4. Poor nutrition

C. Etiology

1. The most likely sources of serious infection in the cancer patient are the respiratory, urinary, and GI tracts.
2. Bacterial infections are the most common.
 - a. Infections with gram-negative organisms (*Pseudomonas aeruginosa*, *Escherichia coli*, *Proteus*, *Klebsiella* spp)
 - b. Incidence of gram-positive infections (*Streptococcus pneumoniae*, *Staphylococcus aureus*, and *Streptococcus epidermidis*) is on the rise and is very common in patients with indwelling venous access devices in some centers.
3. Common viral pathogens include cytomegalovirus, herpes simplex, and herpes zoster.
4. Opportunistic infections (fungal/protozoal) are also seen in these patients but are less common. Gram-negative bacteremia is the most common cause of death in neutropenic cancer patients.

D. Classic clinical scenario

1. The patient appears very ill and may present with fever and hypotension, or the patient appears well and the only indication of occult infection is fever. (Suspect a staphylococcal infection with head and neck tumors).
2. CBC reveals anemia, thrombocytopenia, and granulocytopenia.

E. Diagnostic evaluation

1. CBC with platelet count
2. Urinalysis
3. Chest radiograph
4. Cultures from all appropriate sites

5. Electrolytes
6. Prothrombin time (INR)/partial thromboplastin time

F. Management

1. Emergency therapy with IV fluids and antibiotics is recommended.
2. Because infection may be life-threatening, antibiotics should be started as soon as cultures are drawn. Possible regimens include:
 - a. Monotherapy options: cefepime, ceftazidime, imipenem/cilastatin, meropenem, or piperacillin/tazobactam
 - b. Dual therapy: monotherapy agent and gentamicin, tobramycin, or amikacin
 - c. The above regimens should be modified as follows if specific infections are suspected.
 - (1) Add vancomycin and ceftazidime or cefepime if a gram-positive infection is likely (a clinically obvious catheter related infection, severe mucositis, quinolone prophylaxis), if infection with methicillin-resistant *S aureus* (or *S epidermidis*) is suspected, or if the patient is hypotensive.
 - (2) Add clindamycin or metronidazole if an anaerobic infection is probable (patients with abdominal or gynecologic complaints).
 - d. Consider granulocyte macrophage colony-stimulating factor (GM-CSF) after oncology consult; generally will decrease length of chemotherapy-induced neutropenia by one day (may be important in critically ill patients).

XI. LEPTOMENINGEAL CARCINOMATOSIS

- A. Etiology
 1. Solid tumors in adults (breast, lung, GI carcinomas/melanoma)
 2. Hematogenous malignancies in children
- B. Clinical presentation
 1. Headache is the most common presenting complaint.
 2. Cranial nerve deficits are common.
 3. Spinal symptoms also occur (weakness, numbness, and pain that is frequently radicular in nature).
- C. Diagnostic evaluation
 1. Contrast CT or MRI
 2. Lumbar puncture: CSF ↑ pressure, ↑ protein, ↓ glucose, positive cytology
- D. Emergency department treatment with steroids and osmotherapy (mannitol) is indicated only in the presence of impending herniation.

ONCOLOGIC DISORDERS: PRACTICE CLINICAL SCENARIOS

Answers immediately follow the practice clinical scenarios.

Scenario A

Presentation: A 45-year-old man with colon cancer presents 10 days after starting chemotherapy, with the sole complaint of fever. He denies chest pain, cough, sputum, shortness of breath, upper respiratory symptoms, dysuria or urinary frequency, diarrhea, and vomiting. He has some nausea and decreased oral intake.

Physical examination: Temperature is 102.3°F (39°C), pulse is 110 beats per minute, respiration rate is 16 breaths per minute, and saturation on room air is 98%. Physical examination is unremarkable except for dry mucous membranes.

What is the diagnosis?

Scenario B

Presentation: A 50-year-old woman with breast cancer complains of being tired, weak, anorectic, nauseous, and constipated. Other than breast cancer, past medical history is negative, including for GI disorders or kidney stones.

Physical examination: Temperature is 98.9°F (37.2°C), pulse is 96 beats per minute, respiration rate is 18 breaths per minute, saturation on room air is 96%. Physical examination is unremarkable, including a nontender, nondistended abdomen with normal bowel sounds. Neurologic exam is nonfocal and within normal limits. Her family reports she is a little confused (slow to answer) but is oriented to person, place, and time.

What is the diagnosis?

Scenario C

Presentation: A 65-year-old man with lymphoma presents with back pain that worsens when he lies flat. The pain is located in the mid upper back and is a radicular pain. He also complains of some difficulty with urination but no dysuria.

Physical examination: Temperature is 98.3°F (36.8°C), pulse is 68 beats per minute, respiration rate is 14 breaths per minute, and saturation on room air is 95%. Physical examination reveals tenderness on percussion over the thoracic spine in the area of T9–T10. There is no swelling or skin abnormalities. He has decreased sensation on the left lower leg to light touch and some weakness of the left lower extremity.

What is the diagnosis?

ANSWERS TO PRACTICE CLINICAL SCENARIOS

Scenario A

Diagnosis: fever and neutropenia

Diagnostic evaluation: Chest radiograph and urinalysis are within normal limits. The WBC count is $500/\text{mm}^3$ with 10% neutrophils. Other laboratory studies include a metabolic panel (BUN, creatinine, electrolytes, etc), blood cultures, urine culture, and chest radiograph. The patient is examined for other sources of fever/abscess, including skin (eg, cellulitis), ears, nose, throat, rectal area, etc.

Management: Normal saline is administered IV for mild dehydration. Because the patient has recently been on ciprofloxacin (a quinolone), imipenem and vancomycin are started for broad-spectrum antibiotic coverage, including for methicillin-resistant *Staphylococcus aureus* (MRSA). He is admitted to oncology.

Scenario B

Diagnosis: hypercalcemia

Diagnostic evaluation: A CBC and urinalysis are within normal limits. The calcium concentration is 14 mg/dL. The ECG shows a short QT interval but no dysrhythmias. An abdominal radiograph shows a stone in the renal parenchyma.

Management: The patient is placed on a cardiac monitor and 1 L of normal saline is administered IV immediately. After a second liter of normal saline is administered, she is given 40 mg furosemide and 60 mg pamidronate (both IV) over 3–4 hours while waiting for a bed on the oncology floor.

Scenario C

Diagnosis: acute spinal cord compression

Diagnostic evaluation: Laboratory studies, including a CBC, metabolic panel, and urinalysis, are unremarkable.

Management: A consult with radiation oncology is obtained immediately. The patient is given dexamethasone 100 mg IV, and the diagnosis is confirmed by MRI. His oncologist is notified, and the patient is admitted. A consult with the spine surgeon is obtained.

SYSTEMIC INFECTIOUS DISORDERS

Sepsis and Septic Shock	963
Tetanus.....	964
Malaria.....	966
Toxoplasmosis	967
Rabies	968
Zoonotic Infections.....	969
Rocky Mountain Spotted Fever	969
Lyme Disease.....	971
West Nile Virus.....	973
Biological Warfare Agents.....	974
Smallpox	974
Anthrax	976
Other High-Priority Biologic Agents.....	979
<i>Legionella pneumophila</i>	979
Acquired Immunodeficiency Syndrome (AIDS)	981

SYSTEMIC INFECTIOUS DISORDERS: SELF-ASSESSMENT QUESTIONS

1. Classically, the rash of Rocky Mountain spotted fever:
 - (a) Begins on the wrists and ankles, spreads to the palms and soles, and then progresses centripetally
 - (b) Is coincident with the onset of fever
 - (c) Appears initially at the site of the bite
 - (d) Begins on the chest, face, and neck and spreads to the extremities
2. The vector for spread of Rocky Mountain spotted fever is
 - (a) The male *Ixodes* tick
 - (b) The female *Ixodes* tick
 - (c) The male *Dermacentor* tick
 - (d) The female *Dermacentor* tick
3. All of the following statements regarding erythema migrans are true except:
 - (a) It is tender and painful.
 - (b) It is seen in Stage I.
 - (c) It is warm to the touch.
 - (d) It is nonpruritic.
4. Antibiotics that may be used in treating children with Lyme carditis include all of the following except:
 - (a) Penicillin
 - (b) Tetracycline
 - (c) Amoxicillin
 - (d) Ceftriaxone
5. The animals most commonly associated with the transmission of rabies in the United States are:
 - (a) Rodents, lagomorphs, and cats
 - (b) Skunks, bats, and raccoons
 - (c) Cats, dogs, and foxes
 - (d) Cattle, foxes, and rabbits

6. Characteristic features of anthrax on chest radiograph include all of the following except:
 - (a) Widespread edema
 - (b) Diffuse hemorrhagic lymphadenitis
 - (c) Lobar pneumonia
 - (d) Mediastinal widening
7. Initial antibiotic therapy for inhalational anthrax is:
 - (a) Ciprofloxacin or doxycycline
 - (b) Levofloxacin or a third-generation cephalosporin
 - (c) Ofloxacin or vancomycin
 - (d) Streptomycin or clindamycin
8. All of the following are characteristic of primary HIV except:
 - (a) Positive HIV ELISA
 - (b) Fever
 - (c) Sore throat
 - (d) Increased HIV viral load
9. A minor criteria required for the diagnosis of smallpox is:
 - (a) A febrile prodrome
 - (b) "Belly-button" lesions
 - (c) Lesions at the same stage of evolution
 - (d) Centrifugal distribution of the rash with the greatest concentration of lesions on the face and distal extremities
10. A suspected case of smallpox is one that:
 - (a) Is epidemiologically linked to a laboratory-confirmed case
 - (b) Does not meet the clinical criteria but is clinically consistent with smallpox
 - (c) Is characterized by fever that precedes development of the rash
 - (d) Meets the clinical criteria of smallpox
11. CSF findings in patients with West Nile virus include:
 - (a) PMNs + \uparrow protein + pleocytosis
 - (b) Lymphocytes + \uparrow protein + pleocytosis
 - (c) Lymphocytes + \downarrow protein + pleocytosis
 - (d) PMNs + \downarrow protein + pleocytosis

12. Which of the following statements is true regarding the role of steroids in the treatment of *Pneumocystis jirovecii* (PJP) pneumonia?
- (a) They have no role.
 - (b) They are beneficial as adjunctive therapy in patients with moderate to severe PJP pneumonia.
 - (c) They are beneficial as adjunctive therapy in patients with mild PJP pneumonia.
 - (d) They should be used as a primary therapeutic modality in all patients with PJP pneumonia.
13. Severe malaria is defined by:
- (a) Parasitemia >1%
 - (b) Pulmonary edema
 - (c) Thrombocytosis
 - (d) High fever
14. All of the following features define systemic inflammatory response syndrome (SIRS) except:
- (a) Mean arterial pressure <60 mmHg
 - (b) Heart rate >90 beats per minute
 - (c) Respiratory rate >20 breaths per minute or PaCO₂ <32 mmHg
 - (d) WBC count >12,000/mm³, <4,000/mm³, or >10% bands

ANSWERS

- | | | | |
|------|------|-------|-------|
| 1. a | 5. b | 9. d | 13. b |
| 2. d | 6. c | 10. c | 14. a |
| 3. a | 7. a | 11. b | |
| 4. b | 8. a | 12. b | |

Use the pre-chapter multiple choice question worksheet (page xx) to record and determine the percentage of correct answers for this chapter.

I. SEPSIS AND SEPTIC SHOCK

A. Definitions

1. Systemic inflammatory response syndrome (SIRS) results from an inflammatory cascade of events in response to a noninfectious insult such as an autoimmune disorder, trauma, thromboembolism, or GI bleed. SIRS is defined by two or more of the following:
 - a. Temperature $>101^{\circ}\text{F}$ (38.3°C) or $<96.8^{\circ}\text{F}$ (36°C)
 - b. Heart rate >90 beats per minute
 - c. Respiratory rate >20 breaths per minute or $\text{PaCO}_2 <32$ mmHg
 - d. WBC $>12,000$ cells/mm³, <4000 cells/mm³, or $>10\%$ bands
2. Sepsis = SIRS plus an infection
3. Shock = sepsis + hypotension (mean arterial pressure <60 mmHg)
4. Severe sepsis = sepsis + tissue hypoperfusion and end-organ injury (lactate elevation >2 mmol/L, altered mental status, decreased urine output, disseminated intravascular coagulation, and ARDS)

B. Risk factors

1. Age >65 years old
2. Immunosuppression (AIDS, chronic steroid therapy, chemotherapy, transplant medicines, diabetes)
3. Bacteremia
4. Chronic indwelling lines and tubes (Foley catheters, central lines)

C. Pathogens

1. Gram-positive cocci are most common, followed by gram-negative rods (in the United States).
2. Hospital-acquired pathogens are more likely to be resistant to multiple drugs and result in higher mortality rates.
3. Pathogens from a urinary source result in a lower mortality rate than those from pulmonary, GI, or unknown sources.

D. Diagnostic evaluation

1. History and physical examination for source, such as intra-abdominal infections (cholecystitis, appendicitis, or peritonitis), skin examination (cellulitis, evidence of IV drug abuse), indwelling catheters (dialysis catheter, urinary catheters, central lines), endocarditis
2. Complementary diagnostic studies: chest radiograph, urinalysis, urine culture, blood culture, CBC, serum chemistries, lactate

E. Management

1. Surviving sepsis guidelines: early goal-directed therapy
 - a. Focused on reversing end-organ hypoperfusion/hypoxia by aggressive fluid administration, early broad antibiotic administration, and vasopressor support
 - b. Administration of broad-spectrum antibiotics within 1 hour of recognition of septic shock or severe sepsis
 - c. An initial fluid bolus of 30 mL/kg of normal saline

2. Supplemental oxygen therapy and intubation/mechanical ventilation as indicated by patient's clinical status (increased work of breathing, hypoxia, altered mental status, inability to protect airway)
3. Norepinephrine is vasopressor of first-choice. Epinephrine or vasopressin can be added as needed to maintain mean arterial pressure >65 mmHg.
4. Dopamine is not recommended except in highly select circumstances (eg, patients with low risk of tachyarrhythmias and absolute or relative bradycardia).
5. Corticosteroids are only used to treat adult septic patients with shock if adequate fluid resuscitation and vasopressor therapy are unable to restore hemodynamic stability.
6. Antibiotic therapy
 - a. Community-acquired urinary tract infection: fluoroquinolone or third-generation cephalosporin
 - b. Community-acquired pneumonia: ceftriaxone + macrolide
 - c. Pneumonia, recently hospitalized: fourth-generation cephalosporin or piperacillin/tazobactam + vancomycin
 - d. Unknown source: broad spectrum covering gram-positive, gram-negative and anaerobic organisms with special consideration for methicillin-resistant *Staphylococcus aureus* (MRSA), eg, piperacillin/tazobactam + vancomycin

II. TETANUS

A. Etiology

1. Tetanus is a potentially fatal neuromuscular disease caused by tetanospasmin, which is an exotoxin produced by *Clostridium tetani*, a gram-positive anaerobic rod found chiefly in soil and the feces of many animals (including people).
2. The organism cannot invade healthy tissue, because it requires an anaerobic environment to convert the spores into toxin-producing vegetative forms. The toxin enters peripheral nerve endings and ascends to the spinal cord and brain. The major effect occurs in the spinal cord where the toxin prevents release of the inhibitory neurotransmitters, thereby producing neuromuscular irritability and generalized spasms.
3. High-risk patients
 - a. The elderly (inadequate immunization)
 - b. IV drug abusers
 - c. Patients with decubiti or diabetic ulcers
4. In 10%–20% of cases, no causative wound or injury is ever determined.

B. Clinical presentation

1. The toxin has no effect on the sensorium, and patients are alert and oriented.
2. Trismus ("lockjaw") is the presenting symptom in >50% of cases.
3. The longer the incubation period (range <24 hours to >1 month), the less severe the disease.
4. Four forms of clinical tetanus
 - a. Local tetanus
 - (1) Muscle rigidity close to the site of injury

- (2) Resolves in weeks to months without sequelae
 - (3) May progress to the generalized form
 - b. Generalized tetanus (most common form)
 - (1) Pain and stiffness in the jaw and trunk muscles → facial rigidity (trismus and risus sardonicus) → spasms and tonic contractions → dysphagia, opisthotonos → glottal spasm (with respiratory distress that may lead to arrest) and painful convulsive spasms (arms flexed, fists clenched, legs extended). This may be confused with seizures.
 - (2) Autonomic nervous system dysfunction (occurs during the second week of clinical tetanus) → signs of catecholamine excess (hyperpyrexia, sweating, tachycardia, and labile hypertension) → ↑ blood pressure, pulse, and temperature → high morbidity and mortality rate
 - c. Cephalic tetanus (may occur in fully immunized patients)
 - (1) Head injury or otitis media → cranial nerve dysfunction (cranial nerve VII most common) and trismus
 - (2) Poor prognosis
 - d. Neonatal tetanus
 - (1) Secondary to inadequate maternal immunization
 - (2) Usually results from an infected umbilical stump
 - (3) Extremely high mortality rate
- C. Differential diagnosis
- 1. Strychnine poisoning (strychnine is detectable in the urine)
 - 2. Dystonic reactions to phenothiazines (improves with diphenhydramine)
 - 3. Hypocalcemic tetany
 - 4. Early rabies (unlike tetanus, trismus is uncommon)
- D. Management
- 1. Maintain an adequate airway to prevent asphyxia.
 - 2. Use a benzodiazepine to control muscular spasms.
 - 3. If the patient needs aggressive airway management, neuromuscular blockade with vecuronium. Concomitant sedation is mandatory; use a benzodiazepine, barbiturate, or propofol.
 - 4. Autonomic dysfunction, manifested by sympathetic overactivity, can be treated with labetalol.
 - 5. Meticulous surgical debridement of the causative wound is critical. Theoretically, this decreases continued toxin production.
 - 6. Give human tetanus immune globulin 500 IU IM to neutralize any unbound toxin.
 - 7. The antibiotic of choice is IV metronidazole.
 - 8. Clinical disease does not produce immunity, so give the first dose of tetanus toxoid in the emergency department.
 - 9. A quiet room is essential to prevent precipitation of generalized spasms.

E. Tetanus prophylaxis protocol

Table 42: Tetanus Immunization Protocol

Previous Immunization	Minor Wound	All Other Wounds
Uncertain or <3 doses	Vaccinate	Vaccinate + tetanus immune globulin
≥3 doses with last dose >5 years ago	—	Vaccinate
≥3 doses with last dose >10 years ago	Vaccinate	Vaccinate

1. Complete primary tetanus immunization for adults consists of three IM injections of Td 0.5 mL; the second dose is given 6 weeks after the first, and the third is given 6 months later.
2. Contaminated (tetanus-prone) wounds
 - a. Those contaminated with feces, soil, or saliva
 - b. Punctures, crush wounds, avulsions
 - c. Burns and frostbite
 - d. Those from missiles
 - e. Those >6 hours old or infected on initial presentation
3. Patients >60 years old and immigrants are most likely to have inadequate prior tetanus immunization. If a patient's vaccination status is unknown or uncertain, he or she should receive the complete vaccination series.
4. Give human tetanus immune globulin to individuals with a tetanus-prone wound who have not completed a primary immunization series or who have a contraindication to receiving Td.
5. The CDC recommends that emergency physicians offer tetanus prophylaxis routinely to all patients (regardless of the presenting complaint) who have not been immunized within the last 10 years.

III. MALARIA

A. Etiology

1. *Plasmodium falciparum* (only one that can cause severe disease)
2. *P. malariae*
3. *P. vivax* and *P. ovale* can develop dormant stage for years.
4. Vector: *Anopheles* mosquito

B. Epidemiology

1. Consider malaria in all febrile travelers from areas where malaria is endemic.
2. Incubation 7–30 days (month with inadequate prophylaxis)
3. 1–2.7 million related deaths per year (75% African children)
4. Leading cause of death in children worldwide
5. Leading reason for blood transfusion worldwide

C. Clinical presentation

1. Classic malaria

- a. Cyclic stages of rigors, fever, headache, vomiting, myalgias, seizures (infants), sweats, fatigue
- b. Every 2 days (*P falciparum*, *P vivax*, *P ovale*)
- c. Every 3 days (*P malariae*)
- d. Splenomegaly
- e. Anemia

2. Severe malaria (*P falciparum*)

- a. Severe anemia (parasitemia >5%)
- b. Cerebral malaria: confusion, coma, seizures, permanent deficits (deaf, blind, palsies)
- c. Pulmonary edema, ARDS
- d. Thrombocytopenia
- e. Shock
- f. Acute renal failure

D. Diagnostic evaluation: thin and thick smears

E. Management

- 1. Infectious disease consult (for latest chloroquine sensitivities)
- 2. Chloroquine sensitive (Middle East, Central America west of Panama Canal)
- 3. Chloroquine-resistant strains: quinine plus doxycycline or atovaquone/proguanil
- 4. Severe anemia requires parenteral therapy: quinidine + doxycycline, or artesunate

IV. TOXOPLASMOSIS

A. Etiology

- 1. *Toxoplasma gondii*, ubiquitous protozoan parasite (including cat litter, which should be avoided by pregnant women)
- 2. Immunocompromised and fetus/newborns can develop serious disease with notable CNS (headache, focal neurologic deficits, seizure, altered mental status) and retinal complications (pain and decreased vision).

B. Clinical presentation and diagnostic evaluation

- 1. Retinitis: ophthalmology consult
- 2. CNS: CT imaging with IV contrast and brain biopsy (see AIDS, pages 981–985)

C. Treatment: pyrimethamine and sulfadiazine with folinic acid (to prevent bone-marrow suppression)

V. RABIES

A. Etiology

1. A rhabdoviral infection of the CNS with an incubation period that usually ranges from 30 days to years
2. Extremely rare in the United States (22 cases from 1980 to 1997)
3. Transmitted primarily by the saliva of an infected animal through a break in the victim's skin or mucous membrane.
4. In the United States, ~90% of animal rabies is found only in wildlife (raccoons, skunks, bats, foxes, coyotes, and bobcats). Almost all cases of rabid domestic animals have been reported in rural areas; rabies is prevalent in local wildlife species.
 - a. Major wildlife reservoir is the raccoon, with >60% of all reported cases (most prevalent in the eastern states).
 - b. Skunk rabies is predominant in the central and western states.
 - c. Since 1980, 78% of documented rabies cases have had no history of exposure.

B. Clinical presentation

1. The prodromal period may resemble "viral illness." Symptoms are variable and include fever, headache, malaise, sore throat, dry cough, anorexia, and nausea and vomiting; back pain is common. The patient may not tell you about the bite or the associated tingling, pain, or numbness if other symptoms are more distressing.
2. The excitement phase of clinical presentation is variable and includes thought disturbances with lucid intervals, hypersensitivity to light/sound/touch, agitation, confusion, hallucinations, restlessness, or even seizures.
3. Finally, evidence of brainstem dysfunction becomes apparent. If the patient is lucid, check for diplopia, facial palsies, and dysphagia (which leads to the characteristic hydrophobia). From this point, the patient lapses into a coma. Involvement of the respiratory center follows, and an apneic death ensues.

C. Postexposure prophylactic management

1. **Thoroughly irrigate the wound; this can decrease the amount of virus at the wound site and is an important step in rabies prevention.**
2. **Tetanus prophylaxis as indicated.**
3. **Vaccine selection**
 - a. **Active immunization with human diploid cell vaccine (HDCV) is indicated after a bite from an animal in the suspected group; 1 mL is given IM on day 0 and repeated on days 3, 7, and 14.**
 - b. **Passive immunization with human rabies immune globulin (HRIG) is advised if the animal is (or may be) rabid; 20 IU/kg, with as much as possible infiltrated at the site of the bite, and the remainder given IM at a site near to the wound (avoid gluteal administration because vaccine deposited in fat is poorly immunogenic).**

D. Management

1. Report the incident to the public health department.
2. Domestic animals (cats and dogs)
 - a. If the animal is secured/captured, its behavior is normal, and it remains normal for a 10-day observation period, no treatment is necessary.

- b. If the animal is secured/captured and exhibits abnormal behavior or becomes ill during the 10-day observation period, the animal should be euthanized, the head/brain placed in a plastic bag, and sent refrigerated (not frozen or fixed in formalin) to the state public health department for direct immunofluorescent antibody studies.
 - (1) If laboratory analysis is negative for rabies, no treatment is needed.
 - (2) If laboratory analysis is positive for rabies, treatment with HRIG and the HDVC series is indicated and should be started as soon as possible.
 - c. If the animal cannot be found and captured, public health officials should be contacted regarding the prevalence of rabies in the involved species in your locality and the need for treatment.
 3. Wild animals (skunks, bats, etc): assume they are rabid unless proved negative by direct immunofluorescent antibody testing.
 - a. If the wild animal cannot be found and captured, begin treatment with HRIG and the HDCV series.
 - b. Wild animals that are captured should be tested immediately.
 - (1) If laboratory analysis is negative for rabies, no treatment is needed.
 - (2) If laboratory analysis is positive for rabies, administer HRIG and the HDCV series.
 - c. Consider postexposure prophylaxis for those who were in the same room as a bat and might be unaware of bite or contact.
 4. Bites from rodents, squirrels, and rabbits almost never require postexposure prophylaxis.
- E. Diagnostic evaluation
1. Performed on a specimen of brain tissue obtained postmortem or by biopsy
 2. Fluorescent antibody testing is the procedure of choice (both sensitive and specific).
 3. Staining for Negri bodies: although these intracytoplasmic inclusion bodies are pathognomonic for rabies, they are absent in up to 25% of cases (unacceptably high false-negative rate).

VI. ZOO NOTIC INFECTIONS

A. Rocky Mountain spotted fever (RMSF)

1. Etiology
 - a. Infecting bacteria: the spirochete *Rickettsia rickettsii*
 - b. Vector: the female *Dermacentor* tick (males not infected)
2. Epidemiology
 - a. Two-thirds of patients give a history of tick exposure within the last 14 days (incubation period 2–14 days; the shorter the incubation period, the more serious the disease).
 - b. Peak incidence in spring and summer (when ticks are most active); children 5–9 years old have highest incidence of infection.
 - c. The name RMSF is now a misnomer, because cases have been reported from Canada to Brazil, with highest frequency in the Carolinas, Oklahoma, and Virginia. RMSF is the most common rickettsial disease in the United States and the second most common tickborne disease.

3. Clinical presentation

- a. Fever and myalgia followed by severe headache and GI symptoms precede the appearance of the rash (which usually develops 2–6 days after the onset of fever). Fever is the only universal finding and is usually high ($>102.2^{\circ}\text{F}$ [39°C]).
- b. Although rash is the hallmark of RMSF, it is absent in 5%–15% of cases.
 - (1) An erythematous blanching rash (with 2–3 mm macules) appears initially on the flexor surfaces of the wrists and ankles, spreads to the palms and soles, and then moves rapidly (centrally) to cover most of the body.
 - (2) In the next 2–3 days, the rash becomes maculopapular, darker red, fixed, and finally petechial.
- c. A diagnostic clue on physical examination is extreme tenderness of the gastrocnemius muscle.

4. Diagnostic evaluation (usually useful only retrospectively)

- a. Because serologic tests are often negative in the early phase of illness (results are not usually available during emergency department assessment and availability of specific tests vary from hospital to hospital), the diagnosis of RMSF is a clinical one. The triad of fever, headache, and rash, occurring late spring to early fall, is presumptive evidence for treatment and should not await positive serologic testing. The mortality rate remains high because of delay in starting appropriate antibiotic therapy.
 - (1) Laboratory findings of neutropenia, thrombocytopenia, increased liver function studies, and hyponatremia are suggestive.
 - (2) Indirect fluorescent antibody assay
 - (a) The most sensitive and specific test
 - (b) A titer $>1:64$ is diagnostic
 - (3) Indirect hemagglutination (second most sensitive and specific)
 - (4) The Weil-Felix, complement fixation, and latex agglutination tests are much less sensitive.
- b. Skin biopsy: immunofluorescent antibody staining of a rash specimen
 - (1) 70% sensitivity, 100% specificity
 - (2) The best rapid diagnostic test; if available, it may provide rapid diagnosis (as early as the third day of illness).

5. Management

- a. Early antibiotic therapy significantly reduces the mortality rate so, if you suspect RMSF, start doxycycline therapy.
 - (1) Adults: 100 mg orally or IV bid
 - (2) Children <45 kg: 2.2 mg/kg twice daily
 - (a) Recommended for mild/moderate illness without vomiting
 - (b) Short courses (5–7 days) have not been associated with staining of teeth.
- b. Except for those patients with very mild disease, most require parenteral antibiotics are required.
- c. Therapy should be continued until the patient shows clinical improvement and has been afebrile for 2 days.

6. Prognosis

- a. With appropriate antibiotic therapy, mortality is 5%; in mild cases, recovery is within 20 days.
- b. If untreated, the mortality rate is 8%–20%; pneumonitis is common.

B. Lyme disease

1. Etiology and epidemiology

- a. This tick-borne illness has been reported on every continent in the world except Antarctica; in the United States, the highest frequency is in New England and the middle Atlantic and upper Midwestern states.
- b. The *Ixodes* tick (deer tick) harbors the spirochete *Borrelia burgdorferi*.
- c. The most common tick- and vector-borne disease in the United States; most cases occur between May and August (when tick and human outdoor activity are greatest) and where deer populations are increasing.

2. Clinical presentation

a. Early localized disease (within 1 month)

- (1) History from patients who have been in or near wooded areas
 - (a) Fever and fatigue
 - (b) Malaise and myalgia
 - (c) Headache
- (2) Physical examination: erythema migrans is the hallmark of early Lyme disease → circular skin lesions that begin as a macule or papule at the site of the tick bite and gradually enlarge, at times attaining diameters of ≥ 25 cm. Classically, erythema migrans has a bright-red to blue-red border with a pale interior; it is warm to the touch and is neither tender nor pruritic. Erythema migrans is absent in 20% of cases. *Note:* For pictures of erythema migrans and further specifics on treatment guidelines, see the Centers for Disease Control website (<http://www.cdc.gov/lyme>).

b. Early disseminated disease (weeks to months)

- (1) Neurologic abnormalities predominate the clinical picture.
 - (a) Fluctuating meningoencephalitis (most common)
 - (b) Cranial neuropathy (especially Bell palsy)
 - (c) Peripheral neuropathy (mononeuritis, radiculitis, brachial plexitis)
- (2) Myocarditis is the most common cardiac abnormality; it occurs transiently and is associated with varying degrees of AV block.

c. Late disease (months to years later → chronic infection)

- (1) Migratory oligoarthritis of the large joints is characteristic.
 - (a) The knee is most often affected and may be associated with an effusion.
 - (b) A clue to the diagnosis is the intermittent nature of the joint pain and swelling that occurs for weeks or months; then it completely disappears and recurs sometime later.
- (2) Neurologic symptoms include a wide range of manifestations that may mimic other neurologic conditions and; at this stage of disease, may be the only presenting complaint. Be especially suspicious of any of the following:
 - (a) Subtle encephalopathy (disturbances in mood, memory, sleep) or polyneuropathy (spinal/radicular pain or numbness/tingling in the hands and feet)
 - (b) Cognitive dysfunction (confusion → dementia)
 - (c) Incapacitating fatigue

3. Diagnostic evaluation

- a. Clinical suspicion is of utmost importance, because laboratory testing may be inaccurate or misinterpreted. A history of tick bite (or exposure in an endemic area)

followed by a flu-like syndrome and a rash (erythema migrans) suggests the possibility of Lyme disease. Unfortunately, 66% of patients do not recall a tick bite, and 20%–40% never develop or notice the rash.

- b. Cerebrospinal fluid (CSF) is almost always abnormal but is frequently confused with viral meningitis, because lymphocytes and monocytes are the dominant cell types; however, when associated with an ↑ CSF protein, the diagnosis should be suspected.
 - c. The two-test approach is recommended.
 - (1) Initial positive ELISA screen is followed by Western blot. ELISA has a high false-positive rate but good sensitivity. PCR is not recommended because of poor sensitivity and specificity. There is no value in testing in early localized disease.
 - (2) IgM antibodies develop within 1–2 weeks; IgG antibodies develop within 2–6 weeks. Antibodies may be present for years after Lyme disease treatment.
 - (3) Antibiotics in early Lyme disease may prevent seroconversion.
 - d. ECG for evaluation of AV block
- 4. Post-tick exposure prophylaxis**
- a. Deer tick exposure with tick engorgement for >36 hours is treated with one dose of doxycycline (200 mg) given orally.
 - b. Ticks that are embedded in the skin must be removed ensuring that there is no compression of the gut by using fine tweezers. Grasp the tick as close as possible to the head and pull straight out.
- 5. Management**
- a. Early localized disease: erythema migrans treated with doxycycline or amoxicillin for 10–14 days
 - b. Early disseminated disease
 - (1) Neurologic
 - (a) Isolated facial palsy: oral doxycycline or amoxicillin for 14 days
 - (b) Other neurologic: IV therapy for 14–21 days
 - (2) Cardiac
 - (a) Cardiac monitor and temporary pacing if needed
 - (b) IV therapy until cardiac symptoms resolve up to 21 days
 - c. Late disease
 - (1) Arthritis: 28-day course of oral antibiotics
 - (2) Neurologic: 28-day course of IV antibiotics
 - d. Medication
 - (1) Oral
 - (a) Doxycycline 100 mg twice daily (contraindicated in children <8 years old and in pregnant patients)
 - (b) Amoxicillin 500 mg three times daily
 - (c) Cefuroxime 500 mg twice daily
 - (2) IV
 - (a) Ceftriaxone 2 g once daily
 - (b) Cefotaxime 2 g three times daily
 - e. Jarisch-Herxheimer reaction within 24 hours of antibiotics: host immune response (fever, headache, myalgias) to spirochete antigens seen in up to 15% of cases

6. Admission criteria

- a. All patients with abnormal vital signs
- b. All patients with carditis (monitoring usually required)
- c. Patients who require IV antibiotics
- d. Patients (or family) who are unreliable regarding compliance with medications or instructions
- e. Patients with disabling signs and symptoms in whom the diagnosis is uncertain

C. West Nile virus

1. Overview

- a. A mosquito-borne virus that infects horses, birds, and people; it has been endemic to Asia, Africa, Europe, and Australia before its emergence in North America in 1999. Most patients have only mild symptoms, but occasionally West Nile virus can cause severe meningoencephalitis (usually in older patients).
- b. It is distinguished from other arthropod-borne causes of encephalitis (Eastern and Western equine encephalitis, Japanese and Venezuelan encephalitis) by its geographic distribution, clinical features, and laboratory findings.
 - (1) It is a flavivirus (a subgroup of arboviruses).
 - (2) It is a common cause of viral aseptic meningitis/encephalitis in the Middle East, Asia, and Africa.
 - (3) Cases were initially identified in the United States within the New York metropolitan area and have now spread across the country, including the Western states.

2. Epidemiology

- a. Transmission to horses, birds, and people is via mosquito bites (primarily *Culex pipiens*, the Northern house mosquito).
- b. There is a mosquito-bird-mosquito transmission cycle, with seasonal outbreaks occurring from May to December (based on the life cycle of the mosquito).
- c. Risk factors associated with infection
 - (1) Length of time spent outdoors
 - (2) Failure to apply mosquito repellent
 - (3) Mosquitos seen in the home
- d. Host factors that may increase risk of developing meningoencephalitis
 - (1) Advancing age
 - (2) Hypertension
 - (3) Smoking
 - (4) Cerebrovascular disease

3. Clinical presentation

- a. Most infected patients, including children, are asymptomatic or mildly symptomatic with nonspecific flu-like complaints; meningitis or encephalitis develops in only 1 of every 150 cases.
- b. Patients who develop a neuroinvasive illness present with symptoms that occurred suddenly (fever $>102.2^{\circ}\text{F}$ [39°C], headache, and myalgias).
 - (1) Signs of meningitis: nuchal rigidity

- (2) Symptoms of early encephalitis: mental status changes (confusion, disorientation, ↓ level of consciousness)
 - (3) Signs of severe encephalitis: stupor or coma, some degree of paresis (including flaccid paralysis) or proximal muscle weakness
- 4. Diagnostic evaluation
 - a. CSF: lymphocytes + ↑ protein + pleocytosis (30–100 cells/mm³) and no bacterial pathogens
 - b. Radiologic studies
 - (1) CT rarely shows signs of CNS inflammation.
 - (2) MRI demonstrates enhancement of leptomeninges and/or periventricular areas in 30% of patients.
 - c. Differential diagnosis
 - (1) Herpes simplex virus 1 meningoencephalitis
 - (a) Predominance of PMNs (not lymphocytes), RBCs, and possibly glucose
 - (b) Electroencephalography: temporal lobe focus
 - (2) Bacterial meningitis: positive Gram stain and culture
- 5. Treatment
 - a. Empiric treatment for bacterial meningitis and/or herpes simplex viral encephalitis is appropriate until results of testing for West Nile virus-IgM are clarified.
 - b. Symptomatic and supportive measures are the mainstay of therapy; there is no specific therapy for the treatment of West Nile virus.
- 7. Prevention
 - a. No human vaccine is currently available.
 - b. Reduce mosquito breeding.
 - (1) Drainage of standing water
 - (2) Chemical spraying (to reduce vector transmission)
 - c. Avoid mosquito exposure.

VII. BIOLOGICAL WARFARE AGENTS

A. Smallpox

- 1. Overview
 - a. Variola (smallpox) is an orthopoxvirus. Formerly used as a weapon in the biological warfare program of the Soviet Union, smallpox is one of the most feared potential bioterrorist agents because of the high mortality rate in unvaccinated patients. A single case, anywhere in the world, would constitute a global health emergency.
 - b. Flu-like symptoms (mild to severe) precede the onset of rash by 1–4 days.
 - c. Characteristic clinical features
 - (1) Febrile prodrome (>101°F [38.3°C]) and at least one of the following:
 - (a) Headache
 - (b) Backache
 - (c) Chills

- (d) Vomiting
- (e) Severe abdominal pain
- (f) Prostration
- (2) Smallpox lesions: distinct well-circumscribed vesicles or pustules that may become umbilicated or confluent as they evolve
- (3) High mortality rates (30%) in unvaccinated patients primarily because of lack of specific therapy
- 2. Epidemiology
 - a. There are no known animal or insect vectors. The virus is spread person to person by droplet nuclei or aerosols expelled from the oropharynx; it can also be spread by direct contact (skin lesions or contaminated clothing or bedding).
 - b. Risk of transmission occurs during the prodrome but maximizes with onset of the rash, which usually begins in the mouth and throat and lasts 7–10 days. The risk of infectivity is significantly decreased when the lesions scab over. When the scabs fall off the skin, there is no longer any risk of transmission from the individual, but the scabs remain infectious.
- 3. Clinical presentation (depends on the evolutionary stage of the illness)
 - a. Incubation period (7–17 days): not contagious
 - b. Prodrome (2–4 days): sometimes contagious

Initial symptoms include fever (usually high [101°F ; $38.3^{\circ}\text{--}40^{\circ}\text{C}$]), head/body aches, malaise, \pm vomiting; patients usually feel too sick to carry on normal activities.
 - c. Early rash (~ 4 days): most contagious

Fever and red spots in the mouth \rightarrow spread to face \rightarrow arms and legs \rightarrow hands and feet. Rash develops to bumps with a thick, opaque fluid and that look like a bellybutton (a major distinguishing characteristic of smallpox).
 - d. Pustular rash (~ 5 days): contagious

The bumps become pustules (sharply raised, round, and firm to the touch) often described by patients as a feeling of BB pellets embedded under the skin.
 - e. Pustules and scabs (~ 5 days): contagious

The pustules form a crust and then scab; by the end of the second week (after the appearance of the rash), most of the sores have scabbed over.
 - f. Resolving scabs (~ 6 days): contagious

As the scabs fall off, they leave marks on the skin that eventually become pitted scars; most of the scabs are gone by 3 weeks after the rash appeared.
 - g. Scabs resolved: not contagious

Smallpox remains contagious until all of the scabs have fallen off. The scabs continue to remain infectious.
- 4. Diagnostic evaluation
 - a. Suspicious setting: unexplained fever and a rash in a patient with small red spots or sores on the tongue and buccal mucosa
 - b. Clinical diagnosis
 - (1) Major criteria
 - (a) Febrile prodrome with temperature $>101^{\circ}\text{F}$ (38.3°C)
 - (b) Classic “belly-button” lesions
 - (c) All lesions in the same stage of evolution

(2) Minor criteria

- (a) Centrifugal distribution of the rash on the face and distal extremities
- (b) Lesions appeared first on the oral mucosa and/or palate, face, or arms
- (c) Patient appears toxic or moribund
- (d) Slow evolution of lesions (macules → papules → pustules), each type over a period of 1–2 days
- (e) Lesions on the palms and soles

5. Differential diagnosis

- a. Chickenpox (varicella) is the most likely infectious disease to be confused with smallpox; however, chickenpox has distinctive features that should make the diagnosis easier.
 - (1) Varicella lesions tend to be more superficial, appear in crops, and are present in different stages of development at any given time, ie, papules, vesicles, and scabs all appear together.
 - (2) Varicella lesions tend to be more on the abdomen and back and less on the extremities.
- b. Disseminated herpes zoster is more likely in an immunocompromised host.

6. Specific treatment

- a. Smallpox vaccination: if administered within 4 days of the first exposure, attenuates the course of the illness and protects against a fatal outcome.
- b. Vaccinia immunoglobulin: antibodies against a similar virus may shorten duration of disease.
- c. Postexposure isolation and infection control practices: critical to limiting transmission of smallpox to populations outside the target source.

B. Anthrax**1. Overview**

- a. Inhalational anthrax is a rare and rapidly fatal disease. A potential biological warfare agent, it is often difficult to diagnose early; a high index of suspicion is required.
- b. Clinical presentation: mild flu-like symptoms progress rapidly to respiratory distress → septic shock → multiple organ failure
- c. Characteristic features on chest radiograph
 - (1) Diffuse hemorrhagic lymphadenitis
 - (2) Widespread edema
 - (3) Mediastinal widening
- d. Management
 - (1) Fluoroquinolones
 - (2) Adequate management of respiratory distress and septic/hemorrhagic shock in an ICU setting
 - (3) Vaccination programs
- e. Morbidity/mortality rate is high despite above measures.

2. Epidemiology

- a. The distribution of *Bacillus anthracis* is worldwide and most prevalent among herbivores (cattle, sheep, horses, and goats) that contaminate soil and water holes with *B anthracis* (which may subsequently sporulate and persist in the environment).

- b. While aggressive animal vaccination has lowered the incidence of anthrax among livestock in the United States, the microorganism remains endemic in the soil of Texas, Oklahoma, and the lower Mississippi valley.
 - c. Most human cases of anthrax are due to agricultural or industrial exposure. Shepherds, farmers, and workers in manufacturing plants using infected animal products (particularly contaminated hide, goat hair, wool, or bone) are at highest risk.
 - d. There are three predominant clinical forms of anthrax:
 - (1) Cutaneous anthrax (>95% of cases) from entry of spores through skin abrasions
 - (2) Inhalational anthrax
 - (3) GI anthrax (contaminated meat)
3. Microbiology
- a. *B anthracis* is a large, square-ended, nonmotile, aerobic, gram-positive rod with a centrally located spore.
 - b. On gram stain, the spore appears as an "unstained" area.
 - c. Spores are highly resistant to drying, boiling for 10 minutes, and most disinfectants.
4. Clinical presentation (depends on route of inoculation)
- a. Cutaneous anthrax (most common form; recognition is important because its presence could possibly herald the more serious inhalational anthrax)
 - (1) Through cuts, abrasions, or biting flies, spores of *B anthracis* are introduced into the skin; within hours, they germinate, and vegetative cells multiply and produce anthrax toxin, which causes edema and necrosis.
 - (2) Within 5 days of exposure, small painless (but pruritic) papules appear; 24–48 hours later, the papules enlarge and become vesicular. Edema out of proportion to the size of the vesicle surrounds the lesion and is often associated with fever, malaise, and regional adenopathy.
 - (3) Near the end of the first week, the vesicle usually ruptures, and the remaining ulcer progresses to a black eschar. If recognized and treated promptly, cutaneous anthrax is rarely fatal.
 - b. Pharyngeal and GI anthrax
 - (1) After ingestion of contaminated undercooked meat, pharyngeal ulcers and edema of the neck develop as the anthrax bacilli multiply.
 - (2) After intestinal absorption, bacteria are transported to mesenteric and regional lymph nodes → multiplication and dissemination → hemorrhagic adenitis, ascites, and bacteremia
 - (3) Within 5 days of ingestion of contaminated meat, the patient experiences severe abdominal pain associated with hematemesis and hematochezia. Early diagnosis is difficult, resulting in high mortality.
 - c. Inhalational anthrax
 - (1) Aerosolized anthrax spores >5 μm in size are usually trapped in the upper airway (pharynx, larynx, trachea) or cleared by the mucociliary system.
 - (2) Spores between 2 and 5 μm in size are able to reach the alveolar ducts and alveoli, where they are engulfed by pulmonary macrophages and transported to mediastinal and hilar lymph nodes.
 - (3) After a period of germination, a large amount of anthrax toxin is produced. Regional lymph nodes are quickly overwhelmed, and the toxin finds its way into the systemic circulation.

- (a) Because the organisms are initially transported to mediastinal lymph nodes, a major site of involvement is the mediastinum, which leads to massive hemorrhagic mediastinitis (a typical outcome).
 - (b) Widespread dissemination of the toxin, leading to edema, hemorrhage, necrosis, and septic shock is an ominous sign; death soon follows.
- d. Disease progression
 - (1) Incubation period (up to 6 days): insidious onset of malaise, myalgia, fatigue, nonproductive cough, fever, and an occasional sensation of retrosternal pressure (continues for ~4 days)
 - (2) Sudden onset acute respiratory distress, cyanosis, and hypoxemia. Stridor suggests partial tracheal compression from enlarged mediastinal lymph nodes. The patient may be diaphoretic, with crackles heard on auscultation of the lungs. This stage lasts 24–36 hours and often culminates in death.
 - (a) Chest radiograph: widened mediastinum and pleural effusions are characteristic; lung parenchyma may appear normal.
 - (b) Meningeal involvement: seen in up to 50% of cases; usually bloody and may be associated with subarachnoid hemorrhage
- 5. Diagnosis and treatment of inhalational anthrax
 - a. "Suspicion" is the key to diagnosing and treating inhalational anthrax in the first stage. Guidelines:
 - (1) Do an epidemiologic profile that includes a history of exposure, occupation, and where the victims have been (to identify a possible "hot zone" where anthrax has been found).
 - (2) Has the patient been exposed to someone who has the flu? (Inhalational anthrax is not spread person to person.)
 - (3) Clinical findings suggesting anthrax rather than flu
 - (a) Nausea and vomiting without sore throat, nasal congestion, or rhinorrhea
 - (b) Chest discomfort or pleuritic pain
 - (c) Stuttering, onset of dyspnea, and nonproductive cough
 - (d) Abnormal chest radiograph; consider chest CT if pleural effusion or mediastinal abnormalities are present.
 - (4) Gram stains and cultures should be obtained on blood samples of patients in whom anthrax is suspected; suspicious results should be reported to the CDC for further evaluation.
 - b. Antibiotics and supportive care in an ICU setting are the mainstays of therapy.
 - (1) Consensus recommendations include fluoroquinolones, irrespective of age. Nontoxic victims with cutaneous anthrax can be treated as outpatients with oral ciprofloxacin or doxycycline for 7–10 days. Victims with inhalational, cutaneous, or GI disease and toxicity require IV therapy with ciprofloxacin or doxycycline plus at least two other antibiotics (eg, rifampin, clindamycin, imipenem, or an aminoglycoside).
 - (2) The ICU setting is especially useful for hemodynamic monitoring and management of septic/hemorrhagic shock; also, progressive respiratory insufficiency may necessitate use of ventilatory support.
 - c. People with a credible history of exposure (based on results of forensic investigation by law enforcement agencies) or contact with a known or suspected environment contaminated with *B anthracis* (regardless of laboratory results) should be offered

antimicrobial prophylaxis. Oral ciprofloxacin (500 mg) or doxycycline (100 mg) twice a day is recommended.

- d. For images of cutaneous anthrax and chest radiographs, see <http://en.wikipedia.org/wiki/Anthrax>.

C. Other high-priority biologic agents (can be easily disseminated or transmitted person to person)

1. Pneumonic plague (*Yersinia pestis*)
2. Botulism (*Clostridium botulinum*)
3. Tularemia (*Francisella tularensis*)
4. Viral hemorrhagic fevers (Ebola, Marburg, Lassa, and Argentine viruses)

VIII. LEGIONELLA PNEUMOPHILA

A. Pathophysiology

1. *L. pneumophila* is a gram-negative facultative intracellular bacillus that lives in natural and manmade water systems. It is implicated in as many as 6% of community-acquired pneumonia cases.
2. Transmission occurs via inhalation of contaminated aqueous aerosols from equipment such as cooling towers, evaporative condensers, and shower heads. Person-to-person spread has not been documented.
3. The illness occurs seasonally (summer and fall) and has an incubation period of 2–10 days.
4. Populations at risk
 - a. Patients (particularly men) >50 years old
 - b. Cigarette smokers
 - c. Patients with a significant underlying disease (alcoholism, diabetes mellitus, COPD)
 - d. Immunosuppressed patients (especially those with transplants)
 - e. Patients who live or work near construction or excavation sites
 - f. Recent travel (especially to spas) or changes in plumbing

B. Clinical presentation

1. General systemic manifestations (rigors, high fever, headache, malaise, myalgias, and weakness)
2. Pulmonary symptoms
 - a. An initially dry cough often becomes productive of purulent sputum and is occasionally accompanied by hemoptysis.
 - b. Dyspnea
 - c. Pleuritic chest pain (33%)
 - d. Pontiac fever refers to a milder form of the infection resembling influenza.
3. GI symptoms
 - a. Watery diarrhea (50%)
 - b. Nausea, vomiting, abdominal pain

4. Neurologic signs
 - a. Altered level of consciousness
 - b. Gait disturbance
 - c. Seizures
5. Clinical clues within the past history
 - a. No response to β -lactamase drugs (penicillin, cephalosporins) or aminoglycosides for a recent infection
 - b. Onset of symptoms within 10 days of hospital discharge
6. Physical examination
 - a. Toxic appearance
 - b. Disorientation and confusion
 - c. Diffuse inspiratory rales, progressing to signs of consolidation
 - d. Relative bradycardia (50%)

C. Diagnostic evaluation

1. WBC count 10,000–20,000/mm³ with a left shift
2. Increased sedimentation rate
3. Abnormal serum chemistries
 - a. Increased liver function tests
 - b. Hyponatremia (<130 mEq/L): more common with *Legionella* than with any other cause of pneumonia
4. Proteinuria and microscopic hematuria
5. Gram stain of sputum: PMNs but no predominant organism
6. Chest radiograph
 - a. Unilateral patchy alveolar infiltrate (usually in the lower lobes) progressing to lobar consolidation
 - b. Pleural effusions (16%–33%)
 - c. Cavitary lesions (immunosuppressed patients)
7. Preferred diagnostic tests are the urinary antigen assay and culture of aspiratory secretions in selective media.
8. Direct immunofluorescent antibody staining of sputum, pleural fluid, or lung biopsy has the advantage of providing prompt results but has a sensitivity of only 50%.

D. Treatment

1. Mortality rate is up to 75% without early appropriate antimicrobial therapy and <10% with therapy.
2. The advanced macrolides (especially azithromycin) are preferred by many for treatment of community-acquired pneumonia.
3. Alternative agents
 - a. TMP-SMX and rifampin
 - b. Quinolones (drug of choice for transplant patients)

IX. ACQUIRED IMMUNODEFICIENCY SYNDROME (AIDS)

A. Etiology and pathogenesis

1. Disease complex resulting from an incompetent immune system. It begins when a patient becomes infected with HIV, a retrovirus.
2. Worldwide an estimated 33 million people are HIV positive, and 2.7 million people are newly infected yearly.
3. HIV reduces the number of normally immunocompetent cells (especially T-helper lymphocytes).
4. AIDS is defined by CD4 count <200 cells/mL or presence of AIDS-defining illness.
5. This leads to the development of either Kaposi sarcoma or one of several opportunistic infections (most commonly *Pneumocystis jiroveci* pneumonia, cryptococcal meningitis, or Toxoplasma brain abscess).

B. High-risk populations

1. Homosexuals, bisexuals, and heterosexuals exposed to a partner at risk; presence of other sexually transmitted diseases (eg, chancroid) increases the risk of transmission.
2. IV drug users
3. Prostitutes
4. Patients who underwent tattooing or acupuncture
5. Patients who received blood transfusions between 1978 and 1985
6. Infants born to an HIV-infected mother

C. Most frequent modes of HIV transmission

1. Sexual intercourse
2. Parenteral (IV drug abuse, needle sticks, mucous membrane/skin break exposure)
3. Perinatal maternal-fetal inoculation
4. Breast milk

D. Primary HIV

1. Viral syndrome can be challenging to distinguish.
2. Within 2–4 weeks of transmission, fever, sore throat, nausea, and dry cough develop.
3. Painful, shallow ulcer on the mouth/anus/genitalia (mucous membrane site of HIV transmission).
4. Persistent generalized lymphadenopathy occurs in 11% with early HIV.
5. Seroconversion occurs 10–14 weeks after exposure ($>95\%$ after 6 months).
6. HIV antibody screening with ELISA confirmed by Western blot assay.

E. AIDS-related diseases

1. *Pneumocystis jiroveci* pneumonia (PJP)
 - a. Epidemiology
 - (1) The causative organism is *P jiroveci* (formerly *P carinii*), a genus of unicellular fungi found in the respiratory tract.
 - (2) Seen almost exclusively in immunosuppressed patients
 - (a) Patients with AIDS

- (b) Patients receiving immunosuppressive therapy for cancer (especially corticosteroids) or organ transplantation
 - (c) Premature and malnourished infants
 - (d) Children with primary immunodeficiency disease
- (3) Most common opportunistic infection seen in HIV patients; the leading cause of death in these patients
 - (a) 80% of HIV patients acquire PJP at some time during their illness.
 - (b) It is the initial opportunistic infection in $\geq 60\%$ of those who are not receiving prophylactic therapy.
 - (c) In adults, infection generally does not occur until the CD4 lymphocyte count is < 200 cells/mm³ but may occur with higher counts in children.
- b. Clinical presentation
 - (1) Signs and symptoms develop in a slow and insidious fashion in AIDS patients; most have been symptomatic for 2–3 weeks at the time of diagnosis. Abrupt onset of signs and symptoms with rapid progression occurs more commonly with oncology patients.
 - (2) Patients usually present with dyspnea, nonproductive cough, and fever. Decreased exercise tolerance is also common. Other more variable complaints include weight loss, night sweats, chest pain, fatigue, and chills.
 - (3) Typical physical findings are cyanosis with tachypnea, tachycardia, and a moderately increased temperature.
- c. Diagnostic evaluation
 - (1) Arterial blood gases (or pulse oximetry as substitute) are frequently abnormal.
 - (a) Decreased pO₂
 - (b) Increased alveolar-arterial (A-a) oxygen gradient
 - (c) Low oxygen saturation or desaturation with 10 minutes of exercise
 - (2) Chest radiograph
 - (a) May be normal in up to 20%–30% of patients; this is more common early in the disease process.
 - (b) Classically demonstrates bilateral diffuse interstitial or alveolar infiltrates beginning in the perihilar region and extending in a “bat-wing” pattern.
 - (c) PJP is the most common cause of pneumothorax in patients with AIDS.
 - (3) Lactate dehydrogenase: increased levels in the setting of clinical suspicion and normal chest radiograph are helpful in making the diagnosis. Degree of increase correlates with prognosis.
 - (4) A high-resolution CT scan that reveals patchy nodular densities suggests the diagnosis.
 - (5) The diagnosis in the emergency department is a clinical one. Inpatient evaluation may include bronchoscopy/lavage and sputum monoclonal antibodies.
- d. Treatment
 - (1) Oxygen
 - (2) Antibiotics
 - (a) TMP-SMX
 - i. Initial drug of choice in patients who can tolerate sulfa drugs. It has the advantage of providing coverage for some bacterial pneumonias and is well tolerated in non-AIDS patients.

- ii. Dosage is trimethoprim at 15–20 mg/kg/day and sulfamethoxazole at 75–100 mg/kg/day IV or orally divided in four doses \times 14–21 days.
- iii. Adverse reactions include nausea and vomiting, fever, rash, increased liver enzymes, and neutropenia.

(b) IV Pentamidine

- i. May be used as an alternative drug for patients with a history of severe allergy or adverse reactions to sulfonamides.
- ii. Dosage is 4 mg/kg/day IV over 1 hour \times 14–21 days. Blood pressure must be carefully monitored during infusion, because hypotension is a common adverse effect.
- iii. Adverse reactions include hypotension, syncope, tachycardia, fever, facial flushing, pruritus, renal toxicity, increased liver enzymes, hypoglycemia, rash, thrombocytopenia, neutropenia, pancreatitis, and hallucinations.

(3) Steroids

- (a) **Beneficial as adjunctive therapy in patients with moderate to severe PJP. They limit oxygen deterioration, decrease mortality and respiratory failure, and accelerate recovery.**
- (b) **Administer them to all children and to adult patients with a $pO_2 < 70$ mmHg or a $P(A-a)O_2$ gradient > 35 mmHg.**
- (c) **Start before the antibiotic is given, because hypoxemia may worsen.**
- (d) **Dosage**
 - i. **Prednisone is administered at a starting dosage of 40 mg bid \times 5 days, followed by 40 mg/day \times 5 days, followed by 20 mg/day \times 11 days.**
 - ii. **Methylprednisolone may be substituted for prednisone at 75% of the above dosages if IV therapy is preferred.**

e. Disposition

- (1) Hospitalization is indicated for most patients, especially children and those with a prior history of PJP, because the mortality rate increases with subsequent episodes.
- (2) Patients with mild disease and favorable respiratory parameters can be treated on an outpatient basis if close follow-up can be assured.

f. Prophylaxis

- (1) Recommended for the following patients:
 - (a) Those with a prior episode of PJP pneumonia (the recurrence rate is 60% in AIDS patients)
 - (b) HIV-infected patients with a CD4 count < 200 cells/mL
 - (c) Those undergoing intensive immunosuppressive therapy
- (2) Regimens
 - (a) TMP-SMX 1 double-strength tablet daily
 - (b) Dapsone 50 mg/day
 - (c) Aerosolized pentamidine 300 mg every 4 weeks

2. Cryptococcal meningitis (most common cause of meningitis in HIV patients)

- a. Clinical presentation: headache (with or without nuchal rigidity), confusion (varying degrees), and photophobia; seizures and cranial nerve palsies may also occur.
- b. Diagnostic evaluation
 - (1) Do a CT scan first to exclude an intracranial mass lesion.

- (2) If CT scan is negative, do a lumbar puncture (with opening pressure—will be high) and send the spinal fluid for the usual studies plus:
 - (a) India ink prep (75% sensitive) and/or fungal culture
 - (b) Cryptococcal antigen titer (95% sensitive); the serum cryptococcal antigen titer has the highest sensitivity (98%).
 - c. Management
 - (1) Amphotericin B
 - (2) Infectious disease consult
 3. CNS toxoplasmosis (most common cause of focal encephalitis and the leading cause of intracranial mass lesions in AIDS patients)
 - a. Clinical presentation: fever, headache, focal neurologic signs and symptoms, altered mental status or seizures, and possible chorioretinitis, leading to eye pain and decreased vision
 - b. Diagnostic evaluation: CT scanning with contrast (often shows ring-enhancing lesion) and brain biopsy
 - c. Treatment: pyrimethamine and sulfadiazine with folinic acid (to reduce the incidence of hematologic toxicity)
 4. Kaposi sarcoma
 - a. Seen in 43% of AIDS patients, primarily male homosexuals
 - b. Most common cancer in AIDS patients
 - c. Clinical presentation
 - (1) Reddish brown or bluish red subcutaneous nodules/plaques most commonly found on the face (including the oral cavity), genitalia, and feet
 - (2) Painless, nonpruritic lesions have a spongy texture and range from several millimeters to several centimeters in diameter.
 - (3) Most frequently seen on the skin of the distal extremities
 5. Oral candidiasis (thrush)
 - a. Clinical presentation
 - (1) Sometimes asymptomatic
 - (2) Patients usually complain of a sore or dry mouth, and examination reveals raised white lacy plaques on the tongue and buccal mucosa.
 - (3) The complaint of painful swallowing is suggestive of *Candida* esophagitis, which, if present, confirms the diagnosis of AIDS.
 - b. Diagnostic evaluation: potassium hydroxide prep
 - c. Treatment: clotrimazole troches or a nystatin suspension; oral fluconazole for >3 weeks for esophageal candidiasis
 6. Herpes zoster (shingles)
 - a. Can be a severe and recurrent problem at any stage of HIV infection
 - b. Acyclovir orally (in large doses) or parenterally is the treatment of choice.
 7. Cytomegalovirus retinitis
 - a. Most common ocular complication of AIDS and, if untreated, leads to blindness
 - b. Funduscopy: small, white perivascular infiltrates (early); hemorrhagic necrotizing retinitis (late)
 - c. Treatment with foscarnet or ganciclovir is indicated.
 8. A host of other AIDS-related diseases can affect one or more organ systems, but these are less commonly seen (except for tuberculosis, see pages 323–329). The management of

AIDS and associated opportunistic infections is rapidly evolving; early consult with an infectious disease specialist regarding therapeutic options is highly recommended.

F. Postexposure prophylaxis

1. Risk of HIV transmission: 0.3% for percutaneous inoculation
 - a. Risk greatest if hollow-bore needle in contact with infected blood
 - b. Splashes on mucous membranes or broken skin 0.09% risk of transmission
2. Fluids that can transmit HIV: blood, seminal/vaginal, breast milk (feces, nasal, saliva, sweat, tears, urine, and vomit are not infectious unless bloody)
3. Nonoccupational exposure
 - a. Risk greatest with receptive anal intercourse (1%–30%), insertive anal or receptive vaginal (0.1%–10%), and insertive vaginal (0.1%–1%).
 - b. Risk of needle sharing for IV drug abuse is 0.67% per contact.
4. Management
 - a. Start as soon as possible.
 - b. In general, two reverse transcriptase inhibitors and one protease inhibitor
 - c. 28-day duration unless the source patient is found to be HIV-negative
 - d. 24-hour postexposure prophylaxis hotline (PEpline): (888) 448-4911

Table 43: Recommended HIV Postexposure Prophylaxis for Percutaneous Injuries

Exposure type	Infection Status of Source				
	HIV-positive Class 1 ^a	HIV-positive Class 2 ^a	Source of unknown HIV status ^b	Unknown source ^c	HIV-negative
Less severe ^d	Recommend basic 2-drug PEP	Recommend expanded 3-drug PEP	Generally, no PEP warranted; however, consider basic 2-drug PEP ^e for source with HIV risk factors _f	Generally, no PEP warranted; however, consider basic 2-drug PEP ^e in settings where exposure to HIV-infected persons is likely	No PEP warranted
More severe ^g	Recommend expanded 3-drug PEP	Recommend expanded 3-drug PEP	Generally, no PEP warranted; however, consider basic 2-drug PEP ^e for source with HIV risk factors _f	Generally, no PEP warranted; however, consider basic 2-drug PEP ^e in settings where exposure to HIV-infected persons is likely	No PEP warranted

^a HIV-positive, Class 1: asymptomatic HIV infection or known low viral load (eg, <1,500 copies/mL); HIV-positive, Class 2: symptomatic HIV infection, AIDS, acute seroconversion, or known high viral load. If drug resistance is a concern, obtain expert consultation. Initiation of postexposure prophylaxis (PEP) should not be delayed pending expert consultation, and because expert consultation alone cannot substitute for face-to-face counseling, resources should be available to provide immediate evaluation and follow-up care for all exposures.

^b Source of unknown HIV status (eg, deceased source person with no samples available for HIV testing)

^c Unknown source (eg, a needle from a sharps disposal container)

^d Less severe (eg, solid needle and superficial injury)

^e The designation "consider PEP" indicates that PEP is optional and should be based on an individualized decision between the exposed person and the treating clinician.

^f If PEP is offered and taken and the source is later determined to be HIV-negative, PEP should be discontinued.

^g More severe (eg, large-bore hollow needle, deep puncture, visible blood on device, or needle used in patient's artery or vein)

SYSTEMIC INFECTIOUS DISORDERS: PRACTICE CLINICAL SCENARIOS

Answers immediately follow the practice clinical scenarios.

Scenario A

Presentation: An elderly, demented nursing-home patient presents with altered mental status from baseline (less communicative). The patient is febrile and tachycardic, with a borderline low blood pressure.

What is the diagnosis?

Scenario B

Presentation: An avid hiker presents to the emergency department unable to close one eye or drink water because of a unilateral facial paralysis. She recalls having a rash on her thigh about a month ago that resembled a bull's eye and was not painful or pruritic.

What is the diagnosis?

Scenario C

Presentation: A thin patient with temporal wasting and thrush with a history of IV drug abuse is brought in by concerned family for bizarre behavior. He has had a headache for weeks, and bright light bothers his eyes. Results of laboratory studies show a low absolute lymphocyte count and positive HIV serology. A CT of the brain with and without contrast excludes any intracranial ring-enhancing lesions. A lumbar puncture reveals a high opening pressure and is positive for cryptococcal antigen.

What is the diagnosis?

Scenario D

Presentation: A patient presents with a history of HIV and medication noncompliance. She recalls that she is supposed to take TMP-SMX once per day. She complains of shortness of breath and a nonproductive cough. Her pulse oximetry is in the mid 80s.

What is the diagnosis?

ANSWERS TO PRACTICE CLINICAL SCENARIOS

Scenario A

Diagnosis: severe sepsis

Diagnostic evaluation: Chest radiographs are done, as well as blood and urine cultures, urinalysis, CBC, and lactate.

Management: Treatment includes early and goal-directed therapy with aggressive IV fluid resuscitation, broad-spectrum antibiotics, and admission.

Scenario B

Diagnosis: Lyme disease

Diagnostic evaluation: Diagnostic evaluation involves confirming that there is Lyme disease in the area where the patient has been hiking. Serology should be sent at this stage of the disease. An ECG should be done to screen for cardiac involvement (AV nodal block).

Management: Treatment for isolated facial palsy is doxycycline for 2 weeks. If there is cardiac involvement, IV ceftriaxone is given for up to 3 weeks.

Scenario C

Diagnosis: cryptococcal meningitis

Management: The patient is admitted for IV antifungal therapy and infectious disease consult.

Scenario D

Diagnosis: *Pneumocystis jiroveci* pneumonia

Diagnostic evaluation: Chest radiograph is unremarkable; results of laboratory studies include low WBC and absolute lymphocyte counts, and an increased lactate dehydrogenase level.

Management: The patient is admitted to the hospital, and supplemental oxygen is administered. Steroids are started before TMP-SMX therapy in the emergency department.

NOTES

IMMUNE SYSTEM DISORDERS

RHEUMATOLOGIC DISORDERS.....	993
Raynaud Disease	993
Reiter Syndrome	993
Scleroderma.....	994
Systemic Lupus Erythematosus.....	995
ALLERGIC EMERGENCIES (REACTIONS AND ANAPHYLAXIS)	997
Types of Allergic (Hypersensitivity) Reactions.....	997
Immediate Reactions (Type I).....	997
Cytotoxic Reactions (Type II).....	997
Immune Complex-Mediated Reactions (Type III)	997
Delayed Cell-Mediated Reactions (Type IV)	998
Clinical Manifestations of Allergic (Hypersensitivity) Reactions.....	998
Urticaria	998
Angioedema	999
Erythema Multiforme	1000
Stevens-Johnson Syndrome	1001
Allergic Drug Reactions	1001
Adverse Food Reactions.....	1004
Anaphylaxis.....	1005
Anaphylactoid Reactions	1007

IMMUNE DISORDERS: SELF-ASSESSMENT QUESTIONS

1. The type of reaction that occurs with the tuberculin skin test is most accurately classified as a:
 - (a) Type I allergic reaction
 - (b) Type II allergic reaction
 - (c) Type III allergic reaction
 - (d) Type IV allergic reaction
2. Type I allergic reactions are mediated by:
 - (a) IgE or IgG4
 - (b) IgG or IgM
 - (c) IgM
 - (d) None of the above
3. All of the following statements regarding the skin lesions of erythema multiforme are accurate except:
 - (a) They are typically red, raised, and multishaped with clear centers (target lesions).
 - (b) They may become bullous.
 - (c) They are generally very pruritic.
 - (d) They are classically located on the palms and soles, dorsum of the hands and feet, and on extensor surfaces of the extremities.
4. All of the following statements regarding the Jarisch-Herxheimer reaction are accurate except:
 - (a) It is a febrile reaction to parasitic or bacterial antigens that are liberated when the organisms are destroyed.
 - (b) It is most commonly seen in association with the treatment of spirochetal infections.
 - (c) It generally starts 2–12 hours after the start of antibiotic therapy.
 - (d) It is frequently life threatening.
5. Which of the following reactions requires prior exposure and, therefore, occurs only in sensitized individuals?
 - (a) Phototoxic reactions
 - (b) Photoallergic reactions
 - (c) Anaphylactoid reactions
 - (d) None of the above

6. All of the following agents have been associated with the development of phototoxic reactions except:
- (a) Tetracyclines and sulfas
 - (b) Thiazide diuretics
 - (c) Penicillin and erythromycin
 - (d) Phenothiazines
7. A patient who was stung by a bee approximately 30 minutes before arrival presents with acute respiratory distress and hypotension. On examination, she is wheezing and has perioral and periorbital swelling. Her skin is cold and clammy. The nurses have already placed her on a monitor, given her supplemental oxygen, and started a large-bore IV line. The initial drug therapy of choice is:
- (a) Epinephrine 0.01 mL/kg (up to 0.5 mL of a 1:1,000 solution) SC
 - (b) Benadryl 1–2 mg/kg IV push
 - (c) Inhaled β -adrenergic agent such as albuterol
 - (d) Epinephrine 0.1 mL/kg (up to 5 mL of a 1:10,000 solution) slow IV push
8. All of the following agents have been associated with anaphylactoid reactions except:
- (a) Radiographic contrast media
 - (b) ASA and NSAIDs
 - (c) Acetaminophen
 - (d) Codeine
9. A 40-year-old woman presents with a 5-day history of fever, arthralgias, red and irritated eyes, and rash that has a target appearance on her palms and soles of her feet. Over the last 24 hours, she has had increasing pain in her mouth and has been unable to eat or drink. Physical examination shows bullous lesions on the skin, target lesions on the palms and soles, purulent conjunctivitis, and oral mucosa lesions and desquamation. The most appropriate management is:
- (a) Antipyretics, anti-inflammatories, 7-day course of acyclovir, and outpatient ophthalmology consult
 - (b) IV fluids, steroids, analgesics, and admission for observation
 - (c) Antipyretics, analgesics, 10-day course of doxycycline, and outpatient follow up
 - (d) IV Vancomycin, analgesics, gentamicin ophthalmic drops, and admission for observation
10. Erythema multiforme is commonly induced by all of the following except:
- (a) ACE inhibitors
 - (b) Anticonvulsants
 - (c) Oral hypoglycemics
 - (d) Sulfa drugs

11. Which of the following is not part of the classic triad of systemic lupus erythematosus?
- (a) Fever
 - (b) Joint pain
 - (c) Rash in a female of childbearing age
 - (d) Sulfa drug exposure
12. All of the following are true about scleroderma except:
- (a) It has a strong association with Raynaud phenomenon.
 - (b) It has a strong relationship with Sjögren syndrome.
 - (c) It is predominately a disease affecting the skin and rarely involves other organ systems.
 - (d) Classic presentation includes skin tightness, induration, and pruritus.
13. Which of the following is true about Reiter syndrome?
- (a) Classic triad is conjunctivitis, urethritis, and enteritis.
 - (b) Enteral involvement should be treated with antibiotics.
 - (c) It is associated with *E coli* and *Clostridium difficile* infections.
 - (d) Fever, myalgias, and arthralgias can be safely treated with NSAIDs.
14. An 18-year-old previously healthy woman presents with a complaint of pale and numb finger tips on her left hand after snow skiing for several hours. On physical examination, there is a well-demarcated area of pallor on the distal portion of the second, third, and fourth digits on her left hand. They are numb to the touch. The remainder of the hand is normal. The most appropriate management includes all of the following except:
- (a) Warm the local body part.
 - (b) Evaluate the patient for arterial occlusion.
 - (c) Educate on cessation of vasoconstricting agents such as nicotine.
 - (d) Educate on avoiding precipitating factors.

ANSWERS

- | | | | | |
|------|------|------|-------|-------|
| 1. d | 4. d | 7. d | 10. a | 13. d |
| 2. a | 5. b | 8. c | 11. d | 14. b |
| 3. c | 6. c | 9. b | 12. c | |

Use the pre-chapter multiple choice question worksheet (page xx) to record and determine the percentage of correct answers for this chapter.

RHEUMATOLOGIC DISORDERS

I. RAYNAUD DISEASE

- A. Recurrent artery or arteriole vasospasm most commonly in the fingers and toes, usually in response to stress or cold exposure
- B. Clinical presentation
 - 1. Numbness and paresthesias
 - 2. Pain in the affected area
 - 3. Affected areas show at least two color changes
 - a. Pallor
 - b. Cyanosis
 - c. Hyperemia
 - 4. Transient lasting minutes to hours
- C. Management
 - 1. Warm local body part
 - 2. Discontinue vasoconstricting agents such as nicotine
 - 3. Avoid precipitating factors
 - 4. Calcium channel blockers

II. REITER SYNDROME

- A. **Reactive arthritis following enteric or venereal infections associated with human leukocyte antigen (HLA)-B27**
- B. Associated bacteria
 - 1. *Chlamydia* (most common sexually transmitted infection)
 - 2. *Campylobacter* (most common enteric infection)
 - 3. *Salmonella*
 - 4. *Shigella*
 - 5. *Yersinia*
 - 6. *Cyclospora*
 - 7. Group A streptococci
- C. **Clinical presentation—classic triad: “can’t see, can’t pee, can’t dance with me”**
 - 1. Conjunctivitis—“can’t see”
 - a. Erythema
 - b. Burning
 - c. Tearing
 - d. Pain
 - e. Photophobia

2. Nongonococcal urethritis—"can't pee"
 - a. Frequency
 - b. Dysuria
 - c. Urgency
 - d. Urethral discharge
3. Arthritis—"can't dance with me"
 - a. Asymmetric arthralgia
 - b. Joint stiffness
 - c. Primarily involving the knees, ankles, and feet; worse with rest or inactivity
4. May also have fever, myalgias, and low back pain

D. Management

1. NSAIDs
2. Tetracycline for cases caused by *Chlamydia*
3. Antibiotics are no benefit in cases with enteral causes.

III. SCLERODERMA

A. Systemic autoimmune disease characterized by skin induration and thickening accompanied by tissue fibrosis, chronic inflammatory infiltration of visceral organs, and cellular immune alterations

B. Clinical presentation

1. Skin
 - a. Tightness
 - b. Induration
 - c. Pruritus
2. Vascular
 - a. **95% have associated Raynaud phenomenon**
 - b. Ulcers on fingertips
 - c. Telangiectasis
3. GI
 - a. Gastroesophageal reflux disease
 - b. Dyspepsia
 - c. Constipation alternating with diarrhea
4. Pulmonary
 - a. Dry cough
 - b. Dyspnea
 - c. Pulmonary hypertension
5. Musculoskeletal
 - a. Arthralgia
 - b. Myalgia

- c. Loss of joint range of motion and joint flexion contractures
 - d. Muscle weakness
- 6. Cardiovascular
 - a. Pericardial effusion
 - b. CHF
 - c. Myocardial fibrosis
- 7. ENT
 - a. **Sicca/Sjögren syndrome with dry eyes and dry mouth**
 - b. Poor dentition
 - c. Hoarseness
- 8. Renal
 - a. Hypertension
 - b. Renal insufficiency
- 9. Neurologic
 - a. Trigeminal neuralgia
 - b. Paresthesias and weakness
 - c. Headache
- C. Management
 - 1. **Most treatment focused on complications of the disease**
 - 2. Skin symptoms: phototherapy
 - 3. GI symptoms: antacids, H₂-blockers and proton-pump inhibitors
 - 4. Pulmonary symptoms: cyclophosphamides
 - 5. Renal symptoms: ACE inhibitors
 - 6. Muscle symptoms: low-dose steroids

IV. SYSTEMIC LUPUS ERYTHEMATOSUS

- A. **Chronic autoimmune disease that can affect almost any organ system, making its presentation and course highly variable**
- B. Clinical presentation
 - 1. **Classic triad: fever, joint pain, and rash in a woman of childbearing age**
 - 2. Constitutional: fatigue, fever, weight changes
 - 3. Musculoskeletal: arthralgia, arthropathy, myalgia, avascular necrosis
 - 4. Dermatologic: malar rash, photosensitivity, discoid lupus
 - 5. Renal: acute or chronic renal failure, acute nephritic disease
 - 6. Neuropsychiatric: seizure, psychosis, stroke
 - 7. Pulmonary: pleurisy, pleural effusion, pneumonitis, pulmonary hypertension, interstitial lung disease, pulmonary embolism
 - 8. GI: nausea, dyspepsia, abdominal pain
 - 9. Cardiac: pericarditis, myocarditis, acute MI, tamponade
 - 10. Hematologic: leukopenia, lymphopenia, anemia, or thrombocytopenia

C. Management

1. Emergency department management often focuses on the complications of the disease (acute MI, pulmonary embolism, etc).
2. Antimalarials: hydroxychloroquine
3. Corticosteroids
4. NSAIDs
5. Rheumatology consult

ALLERGIC EMERGENCIES (REACTIONS AND ANAPHYLAXIS)

I. TYPES OF ALLERGIC (HYPERSENSITIVITY) REACTIONS

A. Immediate reactions (Type I)

1. Sequence of events: initial exposure to antigen → activates T-helper lymphocyte itself or binding with protein (hapten-protein complex) → induces plasma cells to produce specific IgE antibody that binds to mast cells and basophils → subsequent reexposure to the antigen → antigen binding with IgE on cell surfaces → release of preformed chemical mediators (histamine, eosinophil chemotactic factor of anaphylaxis [ECF-A], high molecular weight neutrophil chemotactic factor [HMW-NCF], kallikreins) and spontaneously generated mediators (leukotrienes, prostaglandins, platelet-activating factor, thromboxane)
2. These reactions are IgE- or IgG₄-mediated and are responsible for most anaphylactic reactions.
3. Examples
 - a. Drug-induced reactions: penicillin is the most common and the leading cause of fatal anaphylaxis (others include ASA, vancomycin, TMP-SMX, and NSAIDs)
 - b. Hymenoptera venoms
 - c. Foods (shellfish, nuts, egg white, soybeans, wheat, and peanuts [the most common cause of fatal food anaphylaxis])
 - d. Environmental reactions (dust, ragweed, etc)
 - e. Radiologic contrast material
 - f. Food additives (MSG, nitrates/nitrites, tartrazine dyes)

B. Cytotoxic reactions (Type II)

1. Sequence of events: target antigen (a cellular component or a substance that cross-reacts with a cellular component) → complement activation or direct injury to lymphocytes → release of cell mediators
2. These reactions involve binding of IgG or IgM to a cell-bound antigen.
3. Examples
 - a. Blood transfusion reactions
 - b. Immune hemolytic anemias
 - c. Thrombocytopenia (idiopathic thrombocytopenic purpura)

C. Immune complex-mediated reactions (Type III)

1. Sequence of events: soluble antigen-antibody complex → activation of complement system and platelets → IgE complexes and platelet aggregates → release of cell mediators
2. Examples
 - a. Arthus reactions
 - (1) Intradermal injection of an antigen to which the patient has been previously sensitized → edema and erythema at the injection site

- (2) Can occur after administration of tetanus toxoid in the patient who has received too-frequent doses of tetanus toxoid
- b. Serum sickness (fever, rash, arthralgia, and lymphadenopathy)

D. Delayed cell-mediated reactions (Type IV)

1. Sequence of events: antigen-specific T lymphocytes migrate to the site of the antigen injection → release of cell mediators
2. These reactions do not involve antibodies or complement.
3. Signs/symptoms do not develop for 24–72 hours after exposure.
4. Examples: tuberculin skin test, contact dermatitis
5. This classification is somewhat oversimplified in that many allergic reactions consist of more than one type of immune mechanism. Because Types I–III involve complement activation (which can result in varying degrees of anaphylaxis), some degree of overlap does occur. However, the most severe anaphylactic reactions are primarily Type I; Type II is generally less severe, and Type III is the least severe. Type IV does not involve complement activation and therefore does not cause any form of anaphylaxis.

II. CLINICAL MANIFESTATIONS OF ALLERGIC (HYPERSENSITIVITY) REACTIONS

A. Urticaria

1. A vascular reaction involving the skin with transient, pruritic wheals and welts
2. Clinical presentation
 - a. Edematous papules coalescing into plaques that can affect any area of the skin
 - b. White, pink, or erythematous nonscaling lesions that range in size from <1 cm to >10 cm
 - c. Wax and wane over 24–48 hours
3. Causes
 - a. Drug reaction (most common)
 - (1) Penicillin (most commonly implicated) and sulfa
 - (2) Local anesthetics
 - (3) ASA (most commonly implicated)
 - (4) Diuretics
 - (5) Laxatives
 - (6) NSAIDs
 - (7) Morphine and codeine
 - (8) Progesterone
 - b. Infection
 - (1) Infectious mononucleosis
 - (2) Hepatitis B
 - (3) Coxsackie virus
 - (4) Parasitic infestations

- c. Environmental and physical factors
 - (1) Heat or cold
 - (2) Sun exposure
 - (3) Exercise
 - (4) Latex
 - (5) Raw fish
 - (6) Metals (eg, nickel)
 - (7) Animal saliva
- d. Malignancies
- e. Hyperthyroidism
- f. Pregnancy
- g. Foods (fish, eggs, nuts, peanuts, strawberries, lobster)
- h. Mastocytosis (overproliferation of mast cells in tissues)
- 4. Management
 - a. Symptomatic in most cases; however, urticaria alone has been known to progress to full-blown anaphylaxis.
 - b. Diphenhydramine (an H_1 -blocker) is usually the drug of choice for these reactions. Other excellent choices include:
 - (1) Hydroxyzine
 - (2) Chlorpheniramine (pregnancy)
 - (3) Cyproheptadine (cold urticaria)
 - (4) Long-acting nonsedating antihistamine: fexofenadine, loratadine, cetirizine
 - c. The addition of an H_2 -blocker (eg, cimetidine) can provide additional benefit in 10%–15% patients who have an incomplete response to diphenhydramine alone.
 - d. Epinephrine and/or steroid may be required in resistant cases.
 - (1) Moderate to severe cases → methylprednisolone 125 mg IV
 - (2) Severe, life-threatening cases → add IV epinephrine 0.1 mg/kg IV in a 1:10,000 solution
 - e. Patients who are discharged should be:
 - (1) Continued on oral antihistamines for 3 days
 - (2) Continued on steroids if these drugs were required during emergency department treatment
 - (3) Advised to avoid offending agent if known; if unknown, advised to follow up with a dermatologist to identify the offending agent

B. Angioedema

- 1. Edema due to capillary dilation of the deeper layers of skin caused by a C1 esterase inhibitor deficiency or defect that results in decreased levels of C4
- 2. Clinical presentation
 - a. Painless, nonpruritic, nonpitting edema of skin
 - b. Most commonly affects tongue, lips, and face
 - c. Hands and feet
 - d. Scrotum and foreskin
 - e. Can affect abdominal organs and upper airway in more severe cases

3. Drug-induced angioedema

- a. ACE inhibitors (a common cause and can occur any time in course of use, even a year later); NSAIDs, aspirin, contrast media
- b. Management
 - (1) Be ready to establish a definitive airway (fiberoptic intubation procedure of choice).
 - (2) Symptomatic (similar to that for urticaria) in most cases, but progression to anaphylaxis can occur. (Angioedema is not IgE associated; antihistamines and steroids have not clearly been shown to be of benefit.)
 - (3) Administration of fresh frozen plasma to replace C₁ esterase in severe cases
 - (4) Administration of racemic epinephrine should be considered if there is evidence of impending upper airway obstruction.

4. Hereditary angioedema

- a. Edema of the face, airway, or extremities and abdominal pain with nausea, vomiting, and diarrhea is characteristic; hereditary angioedema is caused by C₁-esterase inhibitor deficiency and is usually precipitated by stress or trauma.
- b. Management
 - (1) Frozen plasma (containing C₁ inhibitor)
 - (2) High-dose epinephrine may also be effective. However, life-threatening acute attacks do not respond to standard doses of epinephrine or to antihistamines or steroids.
 - (3) The attenuated androgens (danazol and stanozolol) may help for both acute and chronic hereditary angioedema.

C. Erythema multiforme

1. A more severe variant of an urticarial reaction causing a limited hypersensitivity reaction in the skin
2. Clinical presentation
 - a. Typical lesions are red, raised, and multishaped with clear centers (iris or target lesions) that may become bullous. These nonpruritic lesions have a symmetric distribution and are typically located on the:
 - (1) Palms and soles (characteristic)
 - (2) Dorsum of the hands/wrist (usual location of target lesions) and feet
 - (3) Extensor surfaces of the extremities
 - b. Often following a course of fever, myalgias, headache, and malaise
3. Etiology
 - a. **Drug induced is most common: mnemonic "SOAPS" for Sulfa, Oral hypoglycemic, Anticonvulsants, Penicillin, NSAIDs**
 - b. Infectious agents: herpes simplex virus, Mycoplasma, tuberculosis
 - c. Malignancy: lymphoma
4. Management
 - a. Self-limited
 - b. Treat the underlying condition and remove the offending agent.
 - c. Acyclovir for cases involving herpes simplex virus

D. Stevens-Johnson syndrome

1. A severe, potentially fatal form of erythema multiforme characterized by bullae, mucous membrane lesions, and multisystem involvement
2. Clinical presentation
 - a. Often preceded by a prodrome of malaise, fever, arthralgias, and myalgias
 - b. Purulent conjunctivitis may be so severe that the eyes are swollen shut.
 - c. Mucous membrane breakdown and systemic involvement
3. Precipitating factors
 - a. Long-acting sulfonamides (eg, TMP-SMX)
 - b. Herpes simplex virus and *Mycoplasma* infection
 - c. Drugs: typically antibiotics and anticonvulsants
 - d. Malignancy
4. Management
 - a. Without systemic manifestation and mucous membrane involvement, it can be outpatient with steroid bursts.
 - b. Patients with extensive disease need ICU management, fluids and electrolyte replacement, and management of infectious and thermoregulatory issues.

E. Allergic drug reactions

1. Cutaneous manifestations
 - a. Urticaria with or without angioedema
 - b. Erythema multiforme (or nodosum)
 - c. Anaphylaxis
 - d. Maculopapular eruptions
 - e. Pruritus with or without a rash
 - f. Contact dermatitis
 - g. Photodermatitis
 - h. Purpura
 - i. Fixed drug eruption
 - j. Exfoliative dermatitis
 - k. Stevens-Johnson syndrome
 - l. Toxic epidermal necrolysis
2. Systemic manifestations
 - a. Serum sickness
 - b. Interstitial nephritis and glomerulonephritis
 - c. Vasculitis
 - d. Arthralgias and arthritis
3. Drug reactions associated with cutaneous and systemic manifestations
 - a. Serum sickness
 - (1) Clinical presentation
 - (a) Urticaria
 - (b) Fever and lymphadenopathy
 - (c) Arthralgia and arthritis
 - (d) Glomerulonephritis

- (2) Drug etiology
 - (a) Penicillin and sulfa
 - (b) Barbiturates
 - (c) Thiazide diuretics
- (3) Treatment
 - (a) Remove offending agent.
 - (b) Anti-inflammatories
 - (c) Antihistamines
- b. Acute interstitial nephritis
 - (1) Clinical presentation
 - (a) Fever and skin rash
 - (b) Proteinuria, hematuria, and leukocyturia
 - (c) Eosinophilia and eosinophiluria
 - (d) Azotemia and oliguria
 - (2) Drug etiology
 - (a) Penicillins (especially methicillin)
 - (b) Cephalosporins
 - (c) NSAIDs (especially fenoprofen)
 - (d) Cimetidine
 - (e) Sulfonamides
 - (f) Phenytoin
 - (g) Thiazide diuretics
 - (h) Vancomycin
 - (i) Diltiazem
 - (j) Omeprazole
 - (k) Snake antivenin
 - (l) Streptomycin
 - (3) Management
 - (a) Supportive care measures with fluid and electrolyte maintenance
 - (b) Symptomatic relief of fever and systemic symptoms
 - (c) Avoid nephrotoxic drugs
 - (d) Corticosteroid therapy
- c. Hypersensitivity vasculitis
 - (1) Clinical presentation
 - (a) Palpable purpura on lower extremities
 - (b) Vasculitis involving the CNS, GI tract, lungs, and kidneys
 - (2) Drug etiology
 - (a) Penicillin and sulfa
 - (b) Phenytoin
 - (c) Cephalosporins
 - (d) Thiazide diuretics
 - (e) Allopurinol

- (3) Associated infections (occur in some patients)
 - (a) Chronic bacteremia
 - (b) Hepatitis B and C
 - (c) HIV
- (4) Management
 - (a) Remove offending agent.
 - (b) Systemic corticosteroid therapy
- 4. Other allergic drug reactions
 - a. **Jarisch-Herxheimer reaction**
 - (1) A febrile reaction to parasitic or bacterial antigens that are liberated when the organisms are destroyed by antibiotic therapy
 - (2) Most commonly occurs in association with treatment of spirochetal infections (syphilis, Lyme disease, relapsing fever)
 - (3) Generally occurs 2–12 hours after the start of antibiotic therapy (especially penicillin or a tetracycline) and lasts <48 hours
 - (4) Treatment: supportive care, antipyretics, and occasionally corticosteroids
 - b. Maculopapular eruptions
 - (1) Can be caused by any drug, but ampicillin is an especially common offender
 - (2) If the causative agent is not discontinued, erythroderma or exfoliative dermatitis may develop.
 - c. Contact dermatitis
 - (1) Application of any topical agent (including cosmetic creams and lotions) can cause a cell-mediated reaction (Type IV) characterized by redness, itching, maculopapular, or vesicular eruptions.
 - (2) Treatment consists of topical steroids, antihistamines, and cold wet dressings soaked in Burrow solution.
 - d. Fixed drug eruption
 - (1) A rash that recurs at the same site each time the offending drug is used
 - (2) Lesions are often pigmented (violaceous or dusky red), round or oval in shape, and can be quite pruritic.
 - (3) Most common cause is phenolphthalein in OTC laxatives. Other offenders include:
 - (a) Aspirin
 - (b) Sulfa
 - (c) Tetracycline
 - (d) Barbiturates
 - (e) NSAIDs (especially naproxen and tolmetin)
 - (4) Management consists of discontinuing the offending agent, steroids, and antihistamines.
 - e. Photosensitive reactions
 - (1) Adverse reactions to a combination of a drug (systemic or topically applied) and ultraviolet light
 - (2) Lesions are restricted to sun-exposed areas of the skin (face, nuchal area of the neck, dorsal aspects of the upper extremities).

- (3) **Two types of photosensitive reactions: phototoxic (most common) and photoallergic**
- (a) **A photoallergic reaction is a delayed hypersensitivity reaction. Therefore, it occurs only in sensitized individuals. The eruption is generally intensely pruritic and resembles that seen with contact dermatitis. Topical agents are the most common sensitizers, especially those containing one of the following:**
 - i. **Para-aminobenzoic acid**
 - ii. **Musk ambrette (colognes and perfumes)**
 - iii. **Halogenated salicylanilides (in soaps, cosmetics)**
 - iv. **Phenothiazines**
 - (b) **A phototoxic reaction does not involve immunologic mechanisms and can, therefore, occur on first exposure (drug and adequate sunlight). These reactions appear as exaggerated sunburns; associated pruritus is mild or absent. Commonly implicated drugs include:**
 - i. **Tetracyclines (especially doxycycline)**
 - ii. **Phenothiazines (especially chlorpromazine)**
 - iii. **Sulfonamides and sulfonylureas**
 - iv. **Thiazide diuretics**
 - v. **Chlordiazepoxide**
 - vi. **NSAIDs (especially piroxicam and naproxen)**
- (4) **Management consists of discontinuing the offending agent, avoiding sun exposure, and steroids.**

F. Adverse food reactions — direct histamine release

1. **"Chinese restaurant syndrome": the most common because the food contains most, if not all, of the potential causes (allergens, preservatives, color additives, flavor enhancers, and toxins)**
2. **Sulfites (preservatives)**
 - a. **Can cause flushing, bronchospasm, urticaria, and hypotension within minutes of ingestion; nausea, vomiting, diarrhea, and abdominal cramps can also occur.**
 - b. **Foods containing sulfite: packaged salads, shrimp, dried fruit, gelatin, pickles, sausages, cheeses, wine, and fruit juices**
 - c. **5%–10% of the asthmatic population is sensitive to sulfites, which may produce anaphylactic reactions in these patients.**
3. **Tartrazine dye (yellow dye #5, a food and drug color additive) and parabens (a pharmaceutical additive) can cause urticaria, angioedema, acute bronchospasm, or anaphylaxis.**
4. **Monosodium glutamate (flavor enhancer) can cause flushing, chest pain, wheezing, facial pressure and burning, a burning sensation at the back of the neck, dizziness, paresthesias, headache, palpitations, weakness, nausea, and vomiting.**
5. **Scombroidosis (food toxins) is due to spoiled salt water fish and can cause flushing (resembles a sunburn), headache, urticaria, itching, abdominal cramping, and diarrhea; sometimes a metallic, bitter, or "peppery" taste is noted at the time of ingestion.**

G. Anaphylaxis

1. Pathophysiology

- a. A severe systemic allergic reaction to an antigen that is precipitated by the abrupt release of chemical mediators in a previously sensitized patient and involving two or more organ systems. Prior exposure to the antigen is necessary for anaphylactic shock to occur.
- b. Antigens that can cause anaphylaxis include foods, drugs, vaccines, blood products, pollens, and insect venoms; penicillin remains the leading cause of fatal anaphylaxis.
- c. Patients on β -blockers may have a higher risk of anaphylaxis and of a more severe reaction due to interference with traditional therapies.

2. Clinical presentation

- a. Early signs of impending anaphylaxis
 - (1) Nasal itching or stuffiness
 - (2) A "lump" in the throat (laryngeal edema) or hoarseness
 - (3) Lightheadedness and syncope
 - (4) Chest pain, shortness of breath, and tachypnea
 - (5) Skin complaints: warmth and tingling of the face (especially the mouth), upper chest, and palms or soles are usually the first clinical manifestations of anaphylaxis.
 - (6) GI complaints: nausea, vomiting, diarrhea with tenesmus or crampy abdominal pain
 - (7) An "aura" of impending disaster (may occur in patients with a prior history of anaphylaxis)
- b. Full-blown anaphylaxis
 - (1) Angioedema of the tongue, pharynx, and larynx that can lead quickly to upper airway obstruction
 - (2) Hypotension, tachycardia (or other dysrhythmias), altered sensorium, dizziness, wheezing, and cyanosis that can lead quickly to cardiopulmonary failure. Coughing is an ominous sign that frequently heralds the onset of pulmonary edema.
 - (3) The skin may or may not show the classic wheal-and-flare reaction. If the patient is severely hypotensive, a skin reaction may be difficult to see because of poor perfusion.

3. Management

- a. Supportive measures
 - (1) Supplemental high-flow oxygen and cardiac monitoring
 - (2) Normal saline or lactated Ringer's IV (at least 2 L) for circulatory support
 - (3) Laryngospasm may necessitate oral intubation (the procedure of choice) or cricothyrotomy; racemic epinephrine (2.25% solution 5 mL in 2.5 mL normal saline) may be used as a temporizing measure while setting up for definitive airway management. Sedation and paralysis should be used with caution, because a distorted airway may preclude intubation after paralysis.
- b. Parenteral drug therapy
 - (1) Epinephrine (drug of choice)
 - (a) Mild anaphylaxis (urticaria, rhinitis, conjunctivitis, mild bronchospasm) \rightarrow 0.3–0.5 mL 1:1,000 solution IM
 - (b) Moderate anaphylaxis (generalized urticaria, angioedema, early laryngeal edema, hypotension [blood pressure >80 mmHg]) \rightarrow 0.3–0.5 mL 1:1,000 solution IM. May repeat every 5–20 minutes to a total of 3 doses as needed.

- (c) Severe anaphylaxis (laryngeal edema, respiratory failure, shock) → 1–5 mL 1:10,000 solution IV over 10 minutes. If no improvement, start epinephrine drip as follows: mix 1 mL 1:1,000 solution in 250 mL D5W (4 mcg/mL); run at 1–4 mcg/min. Patients on β -blockers may be refractory to standard dosages of epinephrine; administration of IV glucagon may allow the epinephrine to work.
 - (2) Diphenhydramine
 - (a) Mild anaphylaxis: 25–50 mg orally or IM
 - (b) Moderate anaphylaxis: 50–100 mg IM or IV
 - (c) Severe anaphylaxis: 50–100 mg IV over 3 minutes, may be repeated every 4–6 hours as needed
 - (3) Cimetidine or other H_2 -blockers should be administered IV to those patients whose symptoms persist despite the above therapy. However, if the patient is on a β -blocker, cimetidine should be avoided because it may prolong the activity of the β -blocker.
 - (4) Aerosolized albuterol 0.5 mL (of a 0.5% solution) in 2.5 mL normal saline every 20 minutes as needed should be used to treat persistent bronchospasm. Aerosolized ipratropium with albuterol is also effective; dosage is 0.5 mg in 2.5 mL normal saline every 20 minutes.
 - (5) Corticosteroids
 - (a) Potentiate the effects of epinephrine and decrease capillary permeability, but these effects are not immediate.
 - (b) Initial dose of methylprednisolone is 125–250 mg IV; may be repeated every 6 hours.
 - (6) Glucagon (1 mg IV every 6 minutes as needed) may be beneficial for patients on a β -blocker who have persistent hypotension, as well as for other patients in whom IV epinephrine is relatively contraindicated (eg, pregnant patients, those >35 years old, and those with coronary artery disease) or who are epinephrine-resistant.
 - (7) Heliox (helium oxygen) may be administered to patients with resistant upper airway obstruction or angioedema.
- c. Disposition
- (1) These patients should generally be admitted for observation and repeated doses of antihistamines and steroids; this includes all patients on β -blockers in whom "rebound" can occur once the therapeutic medication effects have dissipated, as well as all other patients whose symptoms were resistant to epinephrine and diphenhydramine therapy.
 - (2) If discharged, these patients should be continued on oral antihistamines and steroids for 3 days and given a prescription for an epinephrine injection kit for initial self-treatment in the event of reexposure.
 - (3) Recommended criteria for discharge
 - (a) Mild anaphylaxis (no hypotension or signs of upper airway obstruction)
 - (b) Rapid and complete response to emergency department therapy
 - (c) 6-hour observation without symptom recurrence
 - (d) Safe discharge to the care of a responsible adult with instructions to return immediately if symptoms recur

H. Anaphylactoid reactions

1. Anaphylactoid reactions resemble anaphylactic reactions but do not require prior exposure; they are not immunologically mediated. The final pathway is identical to that of anaphylaxis but does not require initial sensitizing to the antigen.
2. Commonly implicated agents
 - a. Radiographic contrast media (most common)
 - b. Aspirin
 - c. NSAIDs (especially the pyrazole derivatives)
 - d. Codeine
3. Treatment is the same as that for anaphylaxis.

IMMUNE DISORDERS: PRACTICE CLINICAL SCENARIOS

Answers immediately follow the practice clinical scenarios.

Scenario A

Presentation: A patient presents several days after an uncomplicated illness on TMP-SMX. She has fever, arthralgias, red and irritated eyes, a rash that has a target appearance on her palms and soles of her feet, and oral pain and lesions.

What is the diagnosis?

Scenario B

Presentation: A patient presents with an abrupt onset of swelling of the upper lip and tongue. He denies any difficulty swallowing and shortness of breath. He has no fever, pain, or itching and no history of this in the past. He has a history of hypertension for which he takes a "blood pressure medication" but no other medical problems or allergies.

Physical examination: Heart rate is 86 beats per minute, blood pressure is 124/84 mmHg, respiratory rate is 16 breaths per minute, oxygen saturation is 99%, and temperature is 98.6°F (37°C). Physical examination shows edema and swelling in the upper lip and tongue, which seems to be more notable on the left half of the tongue. The patient is swallowing without difficulties and has a normal neck and breath sounds. The remainder of the dermatologic examination is normal.

What is the diagnosis?

Scenario C

Presentation: A patient presents with a gradual onset of redness, burning, and pain in both eyes. On questioning, patient also notes that he has been suffering from a significant bout of diarrhea and significant pain on urination. He denies any chemical exposure to the eyes and does not recall any injuries. He does not wear contacts or glasses and denies any changes in his ability to see or read.

Physical examination: Heart rate is 104 beats per minute, blood pressure is 118/78 mmHg, respiratory rate is 16 breaths per minute, oxygen saturation is 99%, and temperature is 101°F (38.3°C). Both eyes are erythematous, tearing, and photophobic. Heart and lungs are normal, as is the abdominal examination. The patient seems to have diffuse joint discomfort with range of motion. The remainder of examination is normal.

What is the diagnosis?

Scenario D

Presentation: A patient presents after an abrupt onset of facial flushing, hives, itching, and wheezing while having dinner in "Chinatown." While in the emergency department, he also develops nausea, vomiting, and diffuse abdominal pain.

Physical examination: Heart rate is 110 beats per minute, blood pressure is 90/50 mmHg, respiratory rate is 18 breaths per minute, oxygen saturation is 99%, and temperature is 98.6°F (37°C). The patient's cheeks are flushed, and there is a diffuse urticarial rash on his neck and trunk. Faint end expiratory wheezes are noted on chest auscultation, and the abdomen is soft without peritoneal signs.

What is the diagnosis?

Scenario E

Presentation: A 28-year-old woman presents with fatigue, dyspnea, chest pain, fever, and diffuse arthralgias that have become progressively worse over the last 2 months.

Physical examination: The patient is febrile and has a malar rash. On chest auscultation, there is a cardiac friction rub and decreased breath sounds on the left side. Heart rate is 115 beats per minute, blood pressure is 110/65 mmHg, respiratory rate is 22 breaths per minute, and oxygen saturation is 93%.

What is the diagnosis?

ANSWERS TO PRACTICE CLINICAL SCENARIOS

Scenario A

Diagnosis: Stevens-Johnson syndrome

Diagnostic evaluation: Physical examination findings are consistent with erythema multiforme (target lesions) and associated bullae, mucous membrane lesions and breakdown, and multisystem involvement. The patient may also have a history of herpes simplex virus or Mycoplasma infection. Diagnosis is based on physical examination with the consistent clinical presentation.

Management: These patients should be admitted to the ICU for fluids and electrolyte therapy, and management of infectious and thermoregulatory issues.

Scenario B

Diagnosis: angioedema

Key facts: The patient may also have a family history of such presentation, which would differentiate the drug-induced from the hereditary form of angioedema. The patient would not respond well to treatment with steroids and antihistamines and may develop significant airway issues. In severe cases, epinephrine and fresh frozen plasma may be required.

Scenario C

Diagnosis: Reiter syndrome

Diagnostic evaluation: The patient may also have a history of urethral discharge, a recently treated sexually transmitted infection, or bloody diarrhea representing possible pathogens that may be causing this syndrome. This patient is presenting with the triad of conjunctivitis, urethritis, and arthritis.

Management: In this particular case, an enteral bacterial cause is likely, and the patient would not benefit from antibiotic therapy. Treatment includes NSAIDs and other antipyretics.

Scenario D

Diagnosis: adverse food reaction

Diagnostic evaluation: The patient may also have a history of recently consuming wine, cheese, or other foods that may contain high levels of sulfites. Other possibilities include heavy doses of food dyes, MSG, or fish that potentially could be spoiled.

Management: This direct histamine release reaction due to ingestion of a particular food should be treated with antihistamines and other supportive measures.

Scenario E

Diagnosis: systemic lupus erythematosus

Key facts: The patient could have numerous other presentations suggesting acute emergent complications of the disease involving almost any organ system. This scenario suggests potential pericarditis, pleural effusion, cardiac tamponade, pulmonary embolism, pneumonia, or sepsis. Diagnosis would likely not be formally made in the emergency department but focus on the acute management of the complications of the disease, followed by managing the disease itself with hydroxychloroquine, steroids, NSAIDs, and rheumatology consult.

NOTES

RENAL AND UROLOGIC DISORDERS

Acute Renal Injury/Failure	1017
Chronic Renal Failure.....	1023
Hemodialysis-Related Problems	1025
Complications Associated with Renal Transplantation	1026
Urinary Tract Infections	1027
Nephrolithiasis.....	1030
Male Genital Problems.....	1033
Anatomy	1033
Physical Examination.....	1034
Urinalysis	1034
Common Disorders	1035
Urethritis	1035
Orchitis	1036
Acute Bacterial Prostatitis	1036
Venereal Proctitis.....	1037
Penile Ulcers	1037
Fournier Gangrene.....	1039
Balanoposthitis	1040
Phimosis	1040
Paraphimosis.....	1040
Constriction Injuries to the Penis.....	1041
Fracture of the Penis.....	1041
Peyronie disease	1041
Carcinoma.....	1041
Priapism	1041
Testicular Torsion	1042
Torsion of Appendices Epididymis and Testis.....	1043
Epididymitis.....	1044
Urethral Stricture	1045
Urethral Foreign Bodies	1045
Urinary Retention	1045

RENAL AND UROLOGIC DISORDERS: SELF-ASSESSMENT QUESTIONS

1. A patient presents with acute renal failure. Urinalysis reveals a specific gravity of 1.025 and occasional hyaline casts but is otherwise normal. The urine sodium is <20 , the fractional excretion of sodium (FENa) is $<1\%$, and the renal failure index is $<1\%$. What cause of renal failure does this patient have?
 - (a) Prerenal
 - (b) Renal
 - (c) Postrenal
 - (d) Cannot be determined from the information provided
2. The most common cause of acute intrinsic renal failure is:
 - (a) Glomerulonephritis
 - (b) Malignant hypertension
 - (c) Acute tubular necrosis
 - (d) Vasculitis
3. Complications of acute renal failure include all of the following except:
 - (a) Hyperkalemia
 - (b) Volume overload
 - (c) Hypocalcemia
 - (d) Hypomagnesemia
4. The most sensitive test for detecting rhabdomyolysis is:
 - (a) Urine myoglobin
 - (b) Serum myoglobin
 - (c) Serum creatinine kinase
 - (d) All of the above are equally sensitive.
5. What is the most common cause of life-threatening infection in renal transplant patients?
 - (a) Hepatitis B
 - (b) Toxoplasmosis
 - (c) Cytomegalovirus
 - (d) *Streptococcus pneumoniae*

6. All of the following kidney stones are radiopaque except:
- (a) Cystine
 - (b) Uric acid
 - (c) Magnesium-ammonium-phosphate (struvite)
 - (d) Calcium oxalate
7. All of the following infections may be associated with the development of penile ulcers except:
- (a) Herpes simplex
 - (b) Chancroid
 - (c) Gonorrhea
 - (d) Syphilis
8. A healthy 24-year-old woman presents with a 2-day history of frequency, urgency, and dysuria. She is afebrile, and examination reveals only mild suprapubic tenderness. Urinalysis reveals pyuria and bacteriuria. The most appropriate antibiotic is:
- (a) Amoxicillin and clavulanate
 - (b) Ciprofloxacin
 - (c) Amoxicillin
 - (d) Trimethoprim-sulfamethoxazole
9. A healthy 28-year-old man presents with rectal itching and a discharge. When specifically asked, he states that his sexual preference is male and that he practices anal receptive intercourse. Examination reveals a purulent discharge and inflammation. The most appropriate initial therapy is:
- (a) Ceftriaxone 250 mg IM
 - (b) Doxycycline 100 mg orally bid \times 10 days
 - (c) Ceftriaxone 250 mg IM plus doxycycline 100 mg orally bid \times 7 days
 - (d) Erythromycin base 500 mg orally qid \times 7 days
10. All of the following drug classes are typically associated with urinary retention except:
- (a) Sympatholytics
 - (b) α -Adrenergic stimulants
 - (c) Cyclic antidepressants
 - (d) Antihistamines
11. An elderly man from a nursing home is sent to the emergency department for evaluation of abdominal distention and discomfort. Examination reveals a markedly distended bladder. A Foley catheter is passed and returns 2,000 mL initially and 900 mL/hr over the next 3 hours. The most appropriate management is:
- (a) Discharge to the nursing home with the catheter in place
 - (b) Removal of the catheter and discharge to the nursing home
 - (c) Admission to the hospital for IV fluid resuscitation and correction of electrolyte imbalance
 - (d) None of the above

ANSWERS

- | | |
|------|-------|
| 1. a | 7. c |
| 2. c | 8. d |
| 3. d | 9. c |
| 4. c | 10. a |
| 5. c | 11. c |
| 6. b | |

Use the pre-chapter multiple choice question worksheet (page xx) to record and determine the percentage of correct answers for this chapter.

I. ACUTE RENAL INJURY/FAILURE

A. Causes of acute renal injury/failure according to the underlying pathophysiology

1. Prerenal

a. Occurs when normal kidneys are hypoperfused

- (1) These patients are oliguric (urine volume $<400 \text{ mL/m}^2/\text{day}$).
- (2) In response to volume depletion, the kidney reabsorbs water and salt, producing a concentrated urine (specific gravity 1.020 to 1.030) and a low urine sodium concentration ($<20 \text{ mEq/L}$).
- (3) Because renal parenchymal damage is absent, urine sediment is usually normal with only occasional hyaline casts.

b. Etiology

- (1) Hypovolemia (most common cause)
 - (a) Vomiting and diarrhea
 - (b) Blood loss of any kind
 - (c) Diuretics
 - (d) Skin losses (burns)
- (2) Volume redistribution
 - (a) Sepsis
 - (b) Anaphylaxis
 - (c) Third-space sequestration (pancreatitis, peritonitis, ischemic bowel)
 - (d) Hypoalbuminemic states (cirrhosis, nephrosis)
- (3) Medications that limit glomerular perfusion
 - (a) Prostaglandin-inhibiting drugs (NSAIDs)
 - (b) ACE inhibitors
- (4) Decreased cardiac output
 - (a) CHF/pulmonary edema
 - (b) MI
 - (c) Dysrhythmias
 - (d) Tamponade

2. Postrenal

a. Occurs when there is obstruction of urine flow at any point from the renal collecting system to the urethra

- (1) Urine flow rate varies from anuria (with complete obstruction) to normal urine output (with partial obstruction).
- (2) To produce acute renal failure, ureteral obstruction must be bilateral or occur in a patient who has only one functioning kidney.
- (3) Because the renal parenchyma is not affected, urine sediment may be normal.

b. Etiology

- (1) Ureteral/renal obstruction
 - (a) Stones
 - (b) Blood clots

- (c) Sloughed papillae
- (d) Malignancies (intrinsic or extrinsic)
- (e) Iatrogenic (eg, ligation of ureter during surgery)
- (2) Bladder obstruction
 - (a) Enlarged prostate: benign prostatic hypertrophy or malignancy (most frequent cause)
 - (b) Neurogenic bladder due to drugs, diabetes, or spinal cord injury (another common cause)
 - (c) Carcinoma of the bladder
 - (d) Clot retention
- (3) Urethral obstruction
 - (a) Strictures
 - (b) Phimosis (uncommon cause of azotemia)
 - (c) Meatal stenosis (uncommon cause of azotemia)
 - (d) Posterior urethral valves (a common cause in children)
- 3. Renal intrinsic
 - a. Occurs when there is pathology of the kidney or renal tubule
 - (1) Signs and symptoms (including urine flow) depend on the site and nature of the disorder. The urine sediment is abnormal; exact findings vary with the underlying cause of the renal failure.
 - (a) Acute tubular necrosis: renal tubular casts and muddy brown granular casts
 - (b) Acute interstitial nephritis: eosinophilia, granular and white cell casts
 - (c) Acute glomerulonephritis: RBCs, RBC casts, and proteinuria
 - (2) In these patients, the renal tubules lose their ability to concentrate and reabsorb sodium; therefore, the urine is dilute (specific gravity 1.010), and the urine sodium concentration is increased (>40 mEq/L).
 - b. Etiology
 - (1) Acute tubular necrosis: responsible for 90% of cases and in general is a reversible injury to the renal tubule
 - (a) Ischemia (most common cause of acute tubular necrosis)
 - i. Prolonged hypoperfusion
 - ii. Hemorrhage
 - (b) Nephrotoxins
 - i. Aminoglycosides
 - ii. Heavy metals
 - iii. Contrast
 - (c) Pigments
 - i. Hemoglobin
 - ii. Myoglobin
 - (2) Vascular disease
 - (a) Vasoconstrictive disease (malignant hypertension, thrombocytopenic purpura)
 - (b) Vasculitis
 - (3) Thrombosis (renal artery/vein)

- (4) Acute interstitial nephritis: interstitial inflammation most commonly due to medication
 - (a) Drug-related (penicillin, cephalosporins, sulfonamides, NSAIDs, diuretics, allopurinol, cimetidine, anticoagulants); methicillin is the most commonly implicated drug.
 - (b) Infection-related (bacterial, protozoan, fungal)
 - (c) Immune-related (lupus, leukemia, lymphoma, sarcoidosis)
- (5) Glomerular disease: characterized by inflammation of the glomeruli; seen most commonly in children
 - (a) Postinfectious: poststreptococcal (primary process)
 - (b) Noninfectious: lupus, Goodpasture syndrome (secondary processes)
 - (c) IgA nephropathy: most common cause of primary glomerulonephritis

B. Diagnosis of renal failure according to type

1. The history and physical examination should suggest etiology.
 - a. Hypoperfusion → prerenal
 - b. Signs and symptoms of obstruction → postrenal
 - c. History of a primary disorder that can cause renal disease or a history of exposure to a contrast material, drug, or heavy metal → intrinsic
2. Laboratory studies, procedures, and other diagnostic measures help to identify the cause and site of obstruction due to postrenal causes.
 - a. Postvoid residual urine measurement (by catheter or ultrasound bladder scan)
 - b. Urinalysis
 - c. Renal ultrasound (98% sensitivity in identifying upper-tract obstruction)
 - d. Retrograde pyelography (or antegrade pyelography after placement of percutaneous nephrostomy); intravenous pyelography and other contrast-related procedures are contraindicated in renal failure.
3. Helpful laboratory studies in defining specific abnormalities related to intrinsic renal disease
 - a. Microscopic examination of urine sediment (eosinophilia suggests a drug-related cause)
 - b. Urine for myoglobin
 - c. Urine and serum sodium and creatinine
 - d. Urine osmolality (measures concentrating ability)
4. In cases in which you are not sure of the (acute) type of failure (prerenal, postrenal, or renal), learn to calculate the renal indices.
 - a. These tests are not useful in patients with chronic renal failure, on diuretics, or with a history of obstruction. There can be an overlap between categories, which emphasizes the importance of a good history and physical examination in determining etiology.
 - b. Calculate the indices.

FE_{Na} = fractional excretion of sodium

$$FE_{Na} (\%) = \frac{\text{urine sodium/serum sodium}}{\text{urine creatinine/serum creatinine}} \times 100$$

RFI = renal failure index.

$$\text{RFI (\%)} = \frac{\text{urine sodium}}{\text{urine creatinine/serum creatinine}} \times 100$$

c. Interpret the indices.

Table 44: Laboratory Findings in Renal Failure

	Prerenal	Acute tubular necrosis	Acute glomerular nephritis	Acute interstitial nephritis	Postrenal
Serum BUN/creatinine ratio	>20:1	<20:1	>20:1	<20:1	>20:1
U_{Na} (mEq/L)	<20	>20	<20	Variable	Variable
FE_{Na} (%)	<1	>2	<1	Variable	Variable
Urine osmolality	Increased	<350	Increased	Variable	<350
Urinalysis	Normal or hyaline casts	Granular (muddy brown) casts, renal tubular casts	Dysmorphic RBCs, RBC casts, proteinuria	WBCs, WBC casts, eosinophils	Normal

C. Management

1. Prerenal: correct hypoperfusion and its cause using isotonic fluids.
2. Postrenal: relieve the obstruction temporarily by using a catheter first, then refer the patient for definitive management (which may require other upper tract-related instrumentation, eg, ureteral stent or percutaneous nephrostomy).
3. Renal intrinsic
 - a. Remove the offending agent; treat any specific underlying cause.
 - b. Attempt to increase urine flow in oliguric patients (urine output <400–500 mL/day) and convert them to nonoliguric acute renal failure, which is easier to manage in terms of fluid and electrolyte status using isotonic fluids.
 - (1) This can be accomplished by administering mannitol or furosemide and is successful in one-third to one-half of patients.
 - (2) Dosages
 - (a) Mannitol 12.5–25 g IV
 - (b) Furosemide 2–6 mg/kg IV (maximal dose of 400 mg)
The use of furosemide decreases the need for dialysis and problems associated with fluid overload. However, the ototoxicity of furosemide is proportional to the rate and dosage at which it is given and is high in dosages used in treatment of acute renal injury.
 - (c) Dopamine 1–3 mcg/kg/min IV (increases blood flow, may not improve mortality)
 - c. Support with dialysis if necessary.

D. Complications of acute renal failure

1. Hyperkalemia (most immediate life-threatening complication; see also pages 770–772)
 - a. A serum potassium and ECG are probably two of the most important laboratory studies to obtain in patients with acute renal failure.

- b. Hyperkalemia primarily manifests as cardiotoxicity (cardiac dysrhythmias and sudden death).
- c. Management: determined by the potassium level and the ECG findings and can involve a combination of the following modalities:
 - (1) IV calcium
 - (2) IV glucose and insulin
 - (3) IV sodium bicarbonate
 - (4) IV sodium chloride 3% (in severe hyperkalemia only)
 - (5) Inhaled β -adrenergic agents
 - (6) IV diuretics
 - (7) Cation exchange resin
 - (8) Dialysis
- 2. Hypocalcemia
 - a. Common in chronic renal failure but can occur in acute renal failure in which it is usually asymptomatic
 - b. Treatment: IV calcium gluconate or calcium chloride in symptomatic patients
- 3. Hypermagnesemia is common but rarely significant; magnesium-containing antacids and laxatives should be avoided in these patients.
- 4. High anion gap metabolic acidosis is almost always present but is generally mild and rarely requires correction.
- 5. Volume overload
 - a. Common in oliguric patients
 - b. Can lead to CHF and pulmonary edema
 - c. IV diuretics and nitroglycerin can be used as temporizing measures while arrangements for dialysis are being made.
- E. Antibiotics to avoid in the presence of renal failure
 - 1. Tetracycline
 - 2. Nitrofurantoin
 - 3. Penicillin (most common offender)
 - 4. Cephalosporins and aminoglycosides
 - 5. Bacitracin (uncommonly used)
- F. Indications for dialysis in acute renal failure
 - 1. Hyperkalemia (ECG or clinical manifestations are resistant to therapy)
 - 2. Fluid overload
 - 3. Significant acidosis
 - 4. Presence of certain toxins (ethylene glycol, methanol)
 - 5. Uremic symptoms (pericarditis, encephalopathy, bleeding complications)
 - 6. BUN >100 mg/dL, creatinine >10 mg/dL (relative indication)
- G. Myoglobinuric renal failure
 - 1. Pathophysiology: trauma (especially crush injuries, burns, seizures, prolonged strenuous muscle contraction) \rightarrow rhabdomyolysis (destruction of skeletal muscle cells) \rightarrow release of myoglobin into the serum, which passes through the kidneys and has a direct toxic effect on the renal tubules \rightarrow acute tubular necrosis \rightarrow renal failure (\uparrow serum creatinine)

2. Epidemiology: suspect in the clinical settings listed below
 - a. Major trauma
 - b. Major burns
 - c. Seizures
 - d. Electrical injuries
 - e. Intoxicated patients (especially alcohol and phencyclidine [PCP])
 - (1) In alcoholic patients, rhabdomyolysis is due to the direct toxic effect of ethanol on the skeletal muscle cell membrane in addition to pressure necrosis of muscle as a result of prolonged immobilization.
 - (2) In the phencyclidine (PCP) overdose, rhabdomyolysis is due to the excessive muscle activity and agitation that this drug induces.
 - f. Exertion
 - g. Infections (influenza A and B, coxsackie virus, Epstein-Barr virus, HIV, *Legionella* spp)
3. Diagnostic evaluation
 - a. Urinary findings
 - (1) Color may be smoky (reddish brown) because of the presence of myoglobin (but do not wait for a color change before pursuing the diagnosis).
 - (2) Positive for blood on dipstick examination, but no RBCs are seen on microscopic examination (indicates myoglobinuria).
 - (3) Renal tubular casts and muddy brown granular casts may be present on microscopic examination.
 - (4) Myoglobinuria may be present; however, it is not a reliable marker of rhabdomyolysis, because it is rapidly cleared from the urine and can be absent (despite the presence of significant rhabdomyolysis).
 - b. Serum myoglobin may be positive but, like urine myoglobin, is rapidly cleared and is, therefore, not a reliable indicator of rhabdomyolysis.
 - c. Serum creatine kinase
 - (1) The most sensitive test for detecting rhabdomyolysis
 - (2) Released immediately after muscle injury; clearance from serum is slow.
 - d. Hypocalcemia (63%), hyperkalemia (<40%), and hyperphosphatemia are the most common electrolyte abnormalities that occur in association with rhabdomyolysis and may be significant enough to require treatment.
4. Management
 - a. Avoid using potentially nephrotoxic drugs.
 - (1) NSAIDs
 - (2) Antibiotics (see list on page 1021)
 - b. *Aggressively hydrate* with IV fluids (normal saline or lactated Ringer's) at a rate sufficient to maintain a urine output >2 mL/kg/hr. This is the most important aspect of therapy in these patients.
 - c. Administer mannitol IV at 25 g initially, then add 12.5 g to each subsequent liter of fluid to promote diuresis if fluid hydration alone fails to promote an adequate urine output and clear the pigment.
 - d. Furosemide (2 mg/kg IV) may also be used but is controversial because it may acidify the urine.

- e. Alkalinize the urine with sodium bicarbonate (add 44 mEq to each liter of IV fluid) if hydration alone fails to raise the urine pH to >6.5 (a low urine pH increases the toxicity of myoglobin).
- f. Dialysis may be required to support kidney function until recovery occurs in those who do not respond to the above measures.

H. Contrast-induced nephropathy

1. Defined as an increase in serum creatinine by 0.5 mg/dL or 25% from baseline that occurs 2–3 days after contrast administration
2. Incidence is 1%–2% in the general population, but the relative risk is increased in diabetic patients, the elderly, and those with intrinsic renal disease, CHF, or dehydration.
3. Sodium chloride and sodium bicarbonate hydration, while not risk free, are effective in preventing contrast-induced nephropathy.

II. CHRONIC RENAL FAILURE

A. Pathophysiology and clinical features

1. First stage (\downarrow renal function = $<50\%$ glomerular filtration rate [GFR])
 - a. Renal reserve is lost, but one half of the renal function (GFR) can be gone before serum creatinine increases.
 - b. Excretory and regulatory functions are intact.
2. Second stage (renal insufficiency = 20% – 50% GFR)
 - a. Mild renal failure with some loss of concentrating ability
 - b. Mild anemia
 - c. Sudden fluid loss/gain can precipitate acidosis and severe azotemia.
3. Third stage (renal failure = 5% – 20% GFR)
 - a. Renal failure with loss of concentrating ability
 - b. Severe anemia
 - c. Hypocalcemia, hyperphosphatemia, and hypermagnesemia
 - d. Hyperkalemia (common cause of cardiac arrest in this stage)
4. Fourth stage (uremia = $<5\%$ GFR \rightarrow effects on organ systems)
 - a. Cardiovascular
 - (1) Hypertension
 - (2) Stroke
 - (3) MI
 - (4) CHF
 - (5) Dysrhythmias
 - (6) Pericarditis (uremic and dialysis-induced)
 - b. Hematologic
 - (1) Splenomegaly
 - (2) Anemia with lymphopenia and granulocyte dysfunction
 - (3) Prolonged bleeding time (normal INR, platelet count)

- c. Metabolic
 - (1) Hyperparathyroidism
 - (2) Hyperlipidemia
 - (3) Osteomalacia
 - (4) Goiter
 - (5) Gonadal dysfunction
 - (6) Glucose intolerance
- d. Neurologic
 - (1) Subdural hematoma
 - (2) Peripheral neuropathy
 - (3) Uremic encephalopathy
 - (a) Impaired sensorium
 - (b) Seizures
 - (c) Gait disturbances
 - (d) Slurred speech
 - (e) Asterixis
 - (4) Wernicke encephalopathy
 - (5) "Dialysis dementia" (progressive and fatal)
- e. Immunologic: patients are more susceptible to bacterial, viral, and fungal infections (including tuberculosis).
- f. GI
 - (1) Bleeding
 - (2) Hepatitis
 - (3) Pancreatitis
 - (4) Ascites
- g. Pulmonary
 - (1) Pleural effusions
 - (2) Pulmonary edema

B. Management

- 1. Dialysis
 - a. Peritoneal dialysis
 - (1) Access site (abdominal catheter) is at risk of infection and may lead to peritonitis.
 - (2) Used primarily for treatment of chronic renal failure
 - b. Hemodialysis
 - (1) Access sites (shunts and fistulas) are at risk of infection, clotting, and hemorrhage.
 - (2) Used as a temporary measure for acute renal failure and also for definitive treatment of chronic renal failure
- 2. Renal transplantation
- 3. Acute bleeding: DDAVP (stimulates release of von Willebrand factor from endothelial cells), cryoprecipitate, conjugated estrogens, packed RBCs

III. HEMODIALYSIS-RELATED PROBLEMS

A. Complications associated with vascular access

1. Hemorrhage
 - a. Minor bleeding associated with dialysis puncture or mild trauma
 - (1) Apply nonocclusive pressure to the site. If this is unsuccessful, apply topical thrombin or neutralize excessive anticoagulation with vitamin K or protamine sulfate.
 - (2) Recheck access for presence of a thrill.
 - (3) Evaluate extent of blood loss.
 - (4) Observe in emergency department for rebleeding.
 - b. Extensive bleeding from an aneurysm or pseudoaneurysm
 - (1) Control bleeding with direct pressure.
 - (2) Stabilize patient with IV fluids (normal saline or lactated Ringer's), oxygen, and cardiac monitoring.
 - (3) Draw blood for laboratory studies (CBC, INR, type and cross).
 - (4) Obtain stat vascular surgery consult.
2. Thrombosis (signaled by loss of a thrill in the access)
 - a. Obtain immediate vascular surgery consult to determine whether to remove the clot surgically or use a thrombolytic agent.
 - b. Do not attempt to irrigate the access device, because this can result in clot embolization.
3. Infection (usually due to *Staphylococcus*)
 - a. More common in artificial than native grafts
 - b. Can produce recurrent bacteremia and loss of access site
 - c. Local signs (redness, warmth, tenderness, and induration of access site) may be absent; some patients present with only a fever or recurrent episodes of bacteremia.
 - d. Management
 - (1) Obtain blood cultures.
 - (2) Administer IV antibiotics (usually vancomycin).

B. Dysequilibrium syndrome

1. Occurs during or immediately after dialysis (most frequently during a patient's first dialysis treatment); thought to be caused by cerebral edema, especially if a large volume of fluid has been removed
2. Pathophysiology: dialysis → change in fluid composition and osmolality
3. Clinical presentation: symptoms range from headache, nausea, vomiting, and muscle cramps to altered mental status, grand mal seizures, and coma.
4. Management: symptomatic treatment (rest, analgesics, antiemetics) is all that is needed for most patients; however, other causes of neurologic dysfunction (hypoglycemia, hypocalcemia, intracranial hemorrhage, etc) should be considered and excluded, particularly in patients whose symptoms persist or worsen over a period of observation.

IV. COMPLICATIONS ASSOCIATED WITH RENAL TRANSPLANTATION

A. Graft rejection

1. Types of rejection
 - a. Hyperacute rejection (rarest) occurs within a few minutes to a few hours after surgery and results in irreversible allograft destruction.
 - b. Acute rejection (most common) generally occurs within 1–12 weeks after surgery; 90% may be successfully reversed if recognized and treated promptly.
 - c. Chronic rejection is indolent and difficult to arrest.
2. Clinical presentation
 - a. Tenderness over the allograft (located in the iliac fossa)
 - b. Decreased urine output
 - c. Fever (low-grade)
 - d. Generalized discomfort
 - e. Worsening hypertension
 - f. Precipitous weight gain
 - g. Peripheral edema
3. Diagnostic evaluation should be directed toward excluding other causes of decreased renal function that occur in renal transplant patients (eg, volume contraction, drug-induced nephrotoxicity, infection, etc) and should include:
 - a. Urinalysis
 - b. BUN/creatinine
 - c. Renal ultrasonography and bladder scan for residual urine
 - d. Trough level of cyclosporine
4. Management
 - a. High-dose steroids (IV or orally) for 3–4 days followed by:
 - b. Allograft biopsy (to document the presence of ongoing rejection) in those patients who show little or no improvement, *and*
 - c. Administration of an antibody preparation directed toward lymphocytes (such as OKT3, a monoclonal anti-human T cell antibody [IgG2]) to those patients with documented unremitting allograft rejection.

B. Infections

1. Pathophysiology
 - a. Because of ongoing immunosuppression, infections (particularly opportunistic ones) are common in these patients.
 - b. May present with a paucity of findings (fever and typical physical findings may be absent)
2. Prevention
 - a. In an attempt to prevent these infections, patients are given multiple vaccines and prophylactic medications.
 - b. The regimen generally includes the following:
 - (1) Pneumococcal vaccine

- (2) Hepatitis B vaccine
- (3) Nystatin
- (4) TMP-SMX (for prevention of *Pneumocystis jiroveci* pneumonia)
- 3. Cytomegalovirus infection
 - a. The most common life-threatening infection in these patients (as well as recipients of other solid organ transplants and bone marrow recipients)
 - b. Generally occurs 1–6 months after transplantation surgery
 - c. Manifestations range from daily fever and malaise to leukopenia, lymphadenopathy, ↑ liver function tests, epigastric pain, diarrhea, and pneumonia.
 - d. Evaluation should include a CBC, liver function tests, viral PCR, and chest radiograph.
 - e. Management
 - (1) Reducing immunosuppressive therapy *and*
 - (2) IV ganciclovir (with or without immune globulin) if lung, GI, or liver involvement is present
- 4. Urinary tract infections develop in 20% of patients in the first 4 months after renal transplantation.

V. URINARY TRACT INFECTIONS

- A. Definition: significant bacteriuria in a symptomatic patient
- B. Incidence
 - 1. Females have a steadily increasing incidence with age; peaks occur in infancy, preschool, and childbearing years.
 - 2. Males have approximately the same incidence as females during infancy, but then this rapidly declines and remains low until middle age when prostatism occurs.
- C. Etiology
 - 1. Uncomplicated urinary tract infections (UTIs)
 - a. Represents most UTIs
 - b. Occur in normal individuals in whom no anatomic defect in the urinary system can be found
 - c. Usually caused by gram-negative aerobic bacilli from the gut; *Escherichia coli* is the predominant pathogen.
 - d. In women, a small percentage of these UTIs are caused by *Chlamydia trachomatis* and *Staphylococcus saprophyticus*.
 - 2. Complicated UTIs
 - a. Occur in patients with underlying structural, neurologic, or immunologic disease
 - b. May be caused by unusual organisms (*Pseudomonas*, *Serratia marcescens*, etc); however, typical pathogens predominate.
- D. Normal host-defense mechanisms
 - 1. Acidity: pH < 5.5 discourages bacterial growth.
 - 2. Bladder defense: bladder mucosa destroys the bacteria in the urine remaining on the walls, but this defense mechanism is ineffective if bladder emptying is incomplete.

3. Renal defense: local antibodies, leukocytes, and phagocytes remove bacteria.
4. Urinary hemodynamics: a large urinary flow dilutes bacteria and eliminates them from the system.

E. Clinical presentation

1. Infants: poor feeding, irritability, vomiting
2. Preschoolers: irritability, malaise, dysuria
3. School age: fever (more often in males), dysuria, frequency
4. Adults
 - a. Dysuria, frequency, urgency, and lower abdominal discomfort; patients with upper tract infection may present with lower tract symptoms.
 - b. Low-grade fever, chills, and malaise may be present; a high temperature is more suggestive of pyelonephritis.
 - c. Flank pain and costovertebral angle tenderness can be associated with a simple UTI due to referred pain from the bladder. However, if these findings are present, it is safer to assume that the patient has pyelonephritis. A clue to this diagnosis, if present, is a history of shaking chills.
 - d. Diabetic women who appear ill, presenting with lower abdominal pain with or without a recent history of a UTI, may have emphysematous pyelonephritis. This is a life-threatening bacterial infection that can be quickly diagnosed by bedside ultrasound that shows fluid and gas surrounding the kidney. Nephrectomy may be required.

F. Diagnostic evaluation

1. Urinalysis

- a. A good specimen (no epithelial cells) can be obtained by one of the following methods:
 - (1) Midstream clean catch
 - (2) Catheterization
 - (3) Suprapubic aspiration (newborns and infants)
- b. Microscopic findings
 - (1) Significant pyuria
 - (a) 2–5 WBCs/high-power field on a centrifuged specimen is considered to be significant by most authors.
 - (b) 1–2 WBCs/high-power field in men if bacteria are also present
 - (2) Significant bacteriuria
 - (a) Any bacteria seen on unspun urine is considered significant when viewed under high-powered field.
 - (b) >15/high-powered field (high dry) on spun sediment
- c. Diagnosis of a UTI based on a urinalysis is presumptive.
 - (1) False-positives occur secondary to contamination with bacteria from the skin or from other causes of pyuria.
 - (2) False-negatives occur secondary to diuresis or an infection that is due to a more virulent bacterium (in which case, few organisms are required to cause infection), and they may not be seen in significant numbers on microscopic examination.

2. Definitive diagnosis is made by urine culture.

- a. Traditionally, the presence of a colony count >10⁵/mL was considered consistent with the presence of a UTI. Currently, however, a colony count >10²/mL is considered significant in the symptomatic patient.

b. Cultures should be ordered on:

- (1) Extremes of age (infants, children, and the elderly)
- (2) Adult men
- (3) Pregnant women
- (4) Patients with any of the following:
 - (a) Pyelonephritis
 - (b) Prolonged symptoms (>6 days)
 - (c) Underlying medical disease/immunocompromised (diabetes mellitus, sickle cell anemia, cancer) or history of kidney stones
 - (d) Urologic abnormalities (structural or neurologic)
 - (e) History of recent urologic instrumentation
 - (f) Those who relapse, require hospitalization, or have a chronic indwelling catheter

G. Management

1. In females, look for and exclude cervicitis, pelvic inflammatory disease, and vulvovaginitis.
2. In males, look for and exclude urethritis.
3. Outpatient antibiotic therapy for UTIs
 - a. Patients with uncomplicated UTIs should be treated with an agent effective against *E coli* in community. A 3-day course of antibiotics is optimal if there are no complicating factors; this regimen is inexpensive, associated with good compliance, has fewer adverse effects than a traditional 10–14 day regimen, and has a cure rate similar to that of 7-day therapy.
 - (1) TMP-SMX or amoxicillin-clavulanate (first-line drugs only if local resistance patterns to *E coli* are <10%)
 - (2) Nitrofurantoin
 - (3) A fluoroquinolone is indicated:
 - (a) If local resistance to *E coli* is >10% or
 - (b) If the patient is male with urethritis not caused by a sexually transmitted disease (3-day therapy) or cystitis (7–10 day regimen)
 - b. Pregnant patients: despite increasing resistance rates, ampicillin, amoxicillin, and cephalosporins remain first-line agents.
 - (1) A course of 10–14 days is recommended.
 - (2) Nitrofurantoin is becoming a first-line agent because it is efficacious, inexpensive, and well-tolerated; because of its short half-life, it must be taken for at least 7 days. Do not give to patients with glucose 6-phosphate dehydrogenase deficiency.
 - c. If *Chlamydia trachomatis* is the suspected pathogen (history of new sexual partner or the presence of pyuria without bacteriuria), culture for *Chlamydia trachomatis* and begin therapy with doxycycline, TMP-SMX, or a quinolone (levofloxacin or ofloxacin; ciprofloxacin does not have activity against *Chlamydia*). Therapy should be for 10–14 days.
 - d. Complicated, resistant, or recurrent infections (but no pyelonephritis): start therapy with a quinolone (eg, ciprofloxacin), and adjust as needed based on culture and sensitivity; may require 10–14 days of therapy.
 - e. Single-dose therapy is not appropriate for typical emergency department patients, many of whom have subclinical pyelonephritis and no identified medical doctor.

- (1) Single-dose therapy has not been uniformly accepted in spite of reported cure rates (80%–100%) with TMP-SMX.
- (2) Single-dose therapy should be reserved for the reliable patient with an uncomplicated UTI and an identified personal physician who can reassess the patient within 1 week.
- (3) Single-dose therapy should be avoided in infants and adult men.
- f. Consider offering a 2-day course of a bladder analgesic such as phenazopyridine to patients experiencing painful urination, and encourage frequent voiding.
4. Outpatient antibiotic therapy for patients with mild to moderate uncomplicated pyelonephritis who tolerate oral intake
 - a. Consider "sequential" treatment with a parenteral dose of gentamicin, ceftriaxone, or a fluoroquinolone before starting oral therapy.
 - b. Discharge on a fluoroquinolone for 7–14 days with instructions for follow-up in 1 week.
5. Indications for admission in patients with pyelonephritis
 - a. Severely ill/uroseptic
 - b. The very young or elderly; men >60 years old
 - c. Diagnostic uncertainty
 - d. Underlying anatomic urinary tract abnormality or medical problems (including renal failure)
 - e. History of obstruction, stones, or instrumentation
 - f. Progression of uncomplicated UTI/failed outpatient management
 - g. Persistent vomiting or inability to tolerate oral antibiotics
 - h. Immunocompromised patient (diabetes, cancer, sickle cell disease, transplant recipient)
 - i. Poor social situation/inadequate access to follow up
 - j. Pregnancy
6. Indications for urologic referral to search for an underlying anatomic abnormality
 - a. All children and adult men with UTIs
 - b. Any adult with multiple recurrences of infection (>3/year)
7. Patients with obstruction and a UTI (stones are the most common cause) have a potentially life-threatening condition; a genitourinary consult should be obtained immediately.

VI. NEPHROLITHIASIS

A. Pathophysiology

1. Stones are formed when the urine is supersaturated with a particular mineral (calcium, phosphate, oxalate, urate, etc).
2. Most patients with stones (90%) have a specific underlying metabolic derangement, such as hypercalciuria, hyperparathyroidism, hyperuricosuria, or renal tubular acidosis.

B. Stone composition and etiology

1. Most kidney stones ($\geq 75\%$) contain calcium (oxalate or phosphate) and are radiopaque; they are sometimes caused by chronic hypercalcemic states such as:
 - a. Hyperthyroidism
 - b. Hyperparathyroidism

- c. Neoplasm
 - d. Sarcoidosis
 - e. Multiple myeloma
 - f. Distal renal tubular acidosis
2. Struvite stones (magnesium-ammonium-phosphate) are radiopaque and account for an additional 15% and are caused by a chronic urinary tract infection associated with urea-splitting organisms (usually *Proteus*).
 3. Uric acid stones (6%–10%) are radiolucent and are seen in patients with:
 - a. Gout
 - b. Myeloproliferative disease or leukemia
 - c. High-protein diet
 4. Cystine stones (1%–3%) are radiopaque and associated with a familial trait.

C. Epidemiology

1. Highest incidence is in the southeastern United States during the summer and fall when the intake of oxalate-rich fresh vegetables is greater and dehydration is more common.
2. Patients are typically 20–50 years old.
3. Men, particularly sedentary white men, are affected more than women (ratio 3:1).
4. There is a familial and hereditary predisposition.
5. 90% of stones <5 mm in diameter pass on their own.
6. Recurrence of calculus formation is common; about 50% have a recurrence, and the risk increases with each additional stone episode.

D. Clinical presentation

1. Unilateral colicky pain in the flank, back, or lower quadrant with radiation to the groin, labia, or testicles; distinguishing from an abdominal aortic aneurysm is essential.
2. The pain is usually severe and often accompanied by dysuria, frequency, and hematuria.
3. Nausea and vomiting occur as the pain increases in severity.
4. If the stone passes into the bladder, the severity of pain diminishes markedly but may persist as a dull ache due to ureteral spasm.

E. Diagnostic evaluation

1. Ureteral obstruction is usually due to actual concretions (stones), but it may also be due to crystal aggregates, blood clots, necrotic papillae, or even tumors.
 - a. 90% of "stones" are radiopaque and may be seen on abdominal radiograph.
 - (1) Calcium oxalate
 - (2) Calcium phosphate
 - (3) Magnesium-ammonium-phosphate (struvite)
 - (4) Cystine (moderately radiopaque)
 - b. Radiolucent "stones" can be detected as filling defects on intravenous pyelography.
 - (1) Uric acid
 - (2) Blood clots
 - (3) Sloughed papillae
 - (4) Tumors
 - (5) Xanthine (rare)

- c. Definitive diagnosis of the type of stone requires stone analysis and is indicated for patients in whom stones recur. A 24-hour urine collection and analysis can provide definitive recommendations for prevention of stones; studies include urine volume and pH as well as stone identification (calcium, oxalate, citrate, or magnesium).
 - d. At some point, every patient with a stone should have serum calcium level measured to exclude primary hyperparathyroidism.
2. Laboratory studies
- a. CBC is not indicated but should be considered in the presentation that suggests anemia, infection, or abdominal aortic aneurysm.
 - b. Urinalysis/urine human chorionic gonadotropin (in women of reproductive age)
 - (1) Hematuria is typical of urolithiasis, but its absence does not exclude the diagnosis; 20% of patients will not have microscopic hematuria. Also, there does not appear to be any correlation between the degree of obstruction and the absence of hematuria.
 - (2) If the urinary pH is >7.6 , suspect the presence of urea-splitting organisms (*Proteus*); a pH <7.4 suggests the presence of uric acid calculi and also excludes renal tubular acidosis as the cause.
 - c. Urine culture if infection suspected
 - d. BUN/creatinine (before intravenous pyelography, especially if there is a history of renal disease, diabetes mellitus, or hypertension)
 - e. Serum calcium, phosphorus, and uric acid levels
3. Radiographic studies
- a. CT scanning is the diagnostic study of choice.
 - (1) Spiral or helical CTs have largely replaced intravenous pyelography because they are faster, have greater sensitivity (98%), and do not use contrast material.
 - (2) Both lucent and opaque calculi can be demonstrated as well as hydronephrosis.
 - (3) Identifies other anatomic pathology (eg, abdominal aortic aneurysm, appendicitis, renal carcinoma) but does not provide functional information
 - b. Intravenous pyelography
 - (1) Generally reserved for situations in which a functional or anatomic study is required; should not replace noncontrast CT for initial stone evaluation
 - (2) Findings consistent with the presence of a calculus
 - (a) A delayed nephrogram (delay in appearance of contrast material on the 5-minute film) on the affected side
 - (b) Hydronephrosis/hydroureter
 - (c) Columnization (visualization of the entire ureter on a single film)
 - (d) Extravasation of contrast (uncommon)
 - (3) Occasionally therapeutic; hyperosmolar contrast load \rightarrow \uparrow urine output \rightarrow passage of stone
 - c. Ultrasonography is the initial study of choice in pregnant and pediatric patients in the attempt to limit exposure to ionizing radiation.

F. Management

1. IV hydration with normal saline at a rate sufficient to produce a urine output >2 mL/kg/hr
2. Analgesia

- a. Pain relief has traditionally been achieved with narcotics (for severe pain) and ketorolac (for mild to moderate pain).
- b. However, it now seems clear that ketorolac and narcotics act synergistically with one another. In addition, when used together, the narcotic dosage needed is less (which also reduces adverse effects).
- c. Start with a low dose of an IV narcotic followed by 30 mg of IV ketorolac, then slowly titrate additional doses of IV narcotic until desired pain relief is reached.
- 3. Patients who are discharged should:
 - a. Be encouraged to drink two glasses of water every 2 hours
 - b. Strain their urine to collect the stone for analysis
 - c. Be referred to a urologist for follow-up
 - d. Be advised to return if problems develop (persistent vomiting, fever, chills, unremitting pain)
- 4. Indications for admission
 - a. Presence of an associated urinary tract infection (pyuria, bacteriuria, leukocytosis, fever)
 - b. Uncontrolled pain requiring parenteral narcotics
 - c. Intractable nausea and vomiting
 - d. Hypercalcemic crisis
 - e. Renal insufficiency
 - f. Solitary kidney (relative)
 - g. Intrinsic renal disease (relative)
 - h. High-grade obstruction (relative)
 - i. Stone >5 mm (relative)

VII. MALE GENITAL PROBLEMS

A. Anatomy

- 1. Penis
 - a. Primarily consists of three cylindrical bodies
 - (1) Two corpora cavernosa
 - (a) Form the bulk of the penis and are the major erectile bodies
 - (b) Encased in a thick layer of connective tissue (tunica albuginea)
 - (2) The corpus spongiosum, which surrounds the urethra
 - (3) All three cylinders are covered by Buck fascia.
 - b. Blood supply: internal pudendal artery → deep and superficial penile arteries
 - c. Lymphatic drainage: inguinal nodes
- 2. Scrotum
 - a. Thin skin with inner lining of smooth muscle (dartos)
 - b. Blood supply: branches of the femoral and internal pudendal arteries
 - c. Lymphatic drainage: femoral and inguinal nodes
- 3. Testes
 - a. Average size is 4–5 cm × 3 cm; normal position is upright.

- b. Each testis is encased within a tunica albuginea surrounded by a tunica vaginalis, and there is a small amount of fluid between the two layers; if the fluid increases, a hydrocele results and can be diagnosed by transillumination.
 - c. The testes are anchored to the scrotum at two points: the tunica vaginalis and scrotal ligament; if the tunica vaginalis is capacious and envelops the entire testicle, the testis will not be properly fixed and will usually lie in a horizontal position (rather than upright), which makes it more prone to torsion.
 - d. Blood supply: spermatic arteries (internal and external)
 - e. Lymphatic drainage: iliacs (external, common) and periaortic nodes
 - f. May have an appendix (which is painful with torsion)
4. Epididymis
- a. A single long duct coiled at the superior pole of the testis on the posterior side; within this structure, sperm mature and gain the ability to move.
 - b. May have an appendix (which is painful with torsion)
5. Vas deferens: a muscular tube that extends from the epididymis to the prostate
6. Prostate
- a. This gland surrounds the urethra at a point between the bladder neck and urogenital diaphragm.
 - b. Only the most posterior portion is palpable on rectal examination.

B. Physical examination

1. Penis and scrotum
- a. Inspect the skin for lesions.
 - b. "Milk" the penis to express any discharge to exclude urethritis (which may be asymptomatic).
 - c. Palpate the scrotum for excess fluid (hydrocele) and any abnormalities of the testes and epididymis. Make sure the examining room is warm because, in a cold environment, the testes draw up, making examination difficult.
 - (1) Examine the position of the testes with the patient standing up; palpate for abnormalities with the patient lying down.
 - (2) If a hydrocele is present, refer the patient to a urologist for further evaluation to exclude the presence of an underlying testicular tumor.
 - (3) A noninflamed epididymis feels like an earlobe on palpation.
2. Prostate
- a. On rectal examination, a heart-shaped contour and a "ridge" (median raphe) that distinguishes the two lateral lobes can be palpated.
 - b. A normal prostate feels like the tip of a nose; prostatic carcinoma feels like the tip of a chin.
3. Check for inguinal hernia; in an adult, a direct inguinal hernia is usually acquired.

C. Urinalysis: the "two-cup specimen" technique

- 1. Patient voids 10–15 mL into the first cup (represents urine from the urethra).
- 2. A midstream specimen is also obtained: represents urine from the bladder, kidneys, and prostate.
- 3. These specimens are then examined for the presence of leukocytes.

- a. The presence of significant pyuria in the first cup and fewer WBCs in the midstream cup suggests urethritis.
- b. The presence of equal numbers of WBCs in both specimens suggests infection of the bladder, kidneys, or prostate.

D. Common disorders

1. Urethritis

a. Etiology

- (1) Gonorrhea
- (2) *Chlamydia* (most common cause of non-gonococcal urethritis)
- (3) *Ureaplasma urealyticum* (causes 20%–40% of non-gonococcal cases of urethritis)
- (4) *Trichomonas vaginalis* (responsible for 2%–5% of non-gonococcal cases of urethritis)
- (5) Simultaneous infection (which may be asymptomatic) with both gonorrhea and *Chlamydia* occurs in 30%–50% of cases; antibiotic therapy should target both of these organisms.

b. Classic clinical scenario

- (1) The patient usually complains of dysuria and a urethral discharge but may be asymptomatic.
- (2) Gram stain of the discharge reveals ≥ 5 WBCs/high-power field (oil) and may demonstrate gram-negative intracellular diplococci with gonorrhea; obtain a culture for gonorrhea as well as immunologic preps for *Chlamydia*.

c. Management

- (1) Recommended regimens for gonorrhea include single-dose therapy with one of the following:
 - (a) Ceftriaxone 250 mg IM
 - (b) Cefixime 400 mg orally (no longer recommended because of drug resistance)
 - (c) Ciprofloxacin 500 mg orally (depending on local resistance); no longer recommended by CDC as single-dose therapy
 - (d) Alternative regimens include a single dose of one of the following:
 - i. Spectinomycin 2 g IM (no longer available in United States)
 - ii. Ceftizoxime 500 mg IM
 - iii. Cefotaxime 500 mg IM
 - iv. Cefotetan 1 g IM
 - v. Cefoxitin 2 g IM with probenecid 1 g orally
 - vi. Azithromycin 2 g orally if cephalosporin allergy
- (2) If an associated epididymitis is suspected, 7–10 days of therapy are required.
- (3) A regimen that is effective against *Chlamydia* must also be prescribed. (These chlamydial regimens are also effective against *Ureaplasma urealyticum* in most instances.) Recommended regimens include:
 - (a) Doxycycline 100 mg orally bid \times 7 days or
 - (b) Azithromycin 1 g orally in a single dose
 - (c) Alternative regimens include:
 - i. Erythromycin base 500 mg orally qid \times 7 days or
 - ii. Erythromycin ethylsuccinate 800 mg orally qid \times 7 days or

- iii. Ofloxacin 300 mg orally bid \times 7 days
 - iv. Levofloxacin 500 mg/day orally \times 7 days
 - (3) Infection with *Trichomonas vaginalis*, if present, should be treated with metronidazole 2 g orally. Trichomonal infection should be considered in those patients who present with persistent symptoms (in the absence of noncompliance and reexposure) despite adequate treatment for gonorrhea and *Chlamydia*.
2. Orchitis (inflammation of the testicle)
- a. Isolated orchitis is rare.
 - b. Most cases of orchitis are the result of direct extension of an epididymal infection and occur as a complication of a systemic illness, either bacterial or viral.
 - c. Viral orchitis is most often caused by mumps.
 - d. The patient presents with testicular pain and swelling (urinary and urethral symptoms are usually absent). In the case of mumps orchitis, symptoms usually evolve several days after the onset of parotitis, whereas bacterial orchitis can have a more acute presentation.
 - e. An immediate urologic consult should be obtained to confirm the diagnosis, because orchitis is extremely uncommon; testicular torsion and tumor are far more common.
3. Acute bacterial prostatitis
- a. Etiology
 - (1) Usually bacterial, primarily gram-negative organisms (80% are *E coli*, 20% are *Klebsiella*, *Enterobacter*, *Proteus*, or *Pseudomonas*)
 - (2) Mixed bacterial infections are uncommon.
 - (3) Tuberculosis is a possibility when renal tuberculosis is present.
 - b. Clinical presentation
 - (1) The patient presents with dysuria, frequency, urgency, and occasionally urinary retention accompanied by pain in the perineum, pelvis, and low back; systemic signs of infection may also occur (chills, fever, myalgias, malaise).
 - (2) Examination reveals a tender, swollen prostate that is warm and firm to the touch.
 - (3) Prostatic massage should not be done in patients with acute infection, because it can precipitate bacteremia; urine culture is sufficient for diagnosis, because cystitis is also present in most cases.
 - c. Management
 - (1) Antibiotics
 - (a) Outpatient
 - i. Ofloxacin 300 mg orally bid \times 30 days or
 - ii. Levofloxacin 500 mg/day orally \times 30 days
 - (b) Inpatient (sepsis, urinary retention): gentamicin 3–5 mg/kg/day or tobramycin 3 mg/kg/day plus ampicillin 2 g every 6 hours
 - (2) Prompt urologic consultation is indicated for patients who present with acute urinary retention.
 - (3) Supportive measures
 - (a) Hydration
 - (b) Bed rest
 - (c) Analgesics (NSAIDs or narcotics as needed)
 - (d) Fecal softeners/laxatives (especially if narcotics are prescribed)

4. Venereal proctitis
 - a. Etiology: the most common sexually transmitted pathogens
 - (1) *Neisseria gonorrhoeae*
 - (2) *Chlamydia trachomatis*
 - (3) *Treponema pallidum*
 - (4) Herpes simplex virus (HSV, usually type II)
 - b. Clinical presentation
 - (1) Symptoms range from none (most rectal gonorrhea carriers are asymptomatic) to severe and include rectal itching, burning, pain and/or fullness, tenesmus, and a discharge.
 - (2) Gonorrheal and chlamydial infections are often asymptomatic, but the anal chancre of syphilis and the herpetic lesions of HSV are usually quite painful
 - (3) There is a history of anal intercourse.
 - (4) Rectal examination may be normal but usually reveals inflammation and a purulent discharge; grouped vesicles on an erythematous base or aphthous ulcers may be seen in patients with HSV, whereas a chancre (primary syphilis) or condyloma lata (secondary syphilis) may be seen in patients with syphilis.
 - c. Diagnostic evaluation
 - (1) Perform anoscopy and a Gram stain to confirm the presence of acute proctitis.
 - (2) Exclude HSV infection.
 - (a) Immunofluorescent staining or type-specific viral serologic testing is no longer recommended because of lack of sensitivity.
 - (b) Tzanck smear (multinucleated giant cells)
 - (3) Perform bacterial and viral cultures.
 - (4) Send blood to check for syphilis (VDRL).
 - d. Management
 - (1) Ceftriaxone 250 mg IM once plus doxycycline 100 mg orally bid \times 7 days is the treatment of choice.
 - (2) Alternatives to the above regimen are the same as those listed for gonococcal urethritis.
 - (3) If herpetic or syphilitic lesions are present, appropriate therapy for these infections should also be given (described below in the section on penile lesions).
 - (4) Refer all patients for appropriate follow-up.
5. Penile ulcers
 - a. Etiology
 - (1) HSV (types I and II)
 - (2) Syphilis
 - (3) Chancroid (*Haemophilus ducreyi*)
 - (4) Granuloma inguinale (*Klebsiella granulomatis* [formerly *Calymmatobacterium granulomatis*])
 - (5) Lymphogranuloma venereum (*Chlamydia trachomatis*)
 - (6) Behcet syndrome
 - (a) Vasculitis of uncertain etiology but thought to be an autoimmune disorder
 - (b) Clinical triad

- i. Chronic oral ulcerations
 - ii. Relapsing iridocyclitis
 - iii. Genital ulcers
- (c) Can be complicated by polyarthritides and erythema nodosum
- (d) Lasts for many years with relapses and remissions but can be suppressed by corticosteroids.
- b. Diagnostic evaluation
 - (1) Serologic testing for syphilis: a VDRL or rapid plasma reagent (nontreponemal tests) should be done initially and, if positive, should be confirmed with an FTA-ABS (a treponemal test). A VDRL should also be repeated at 3, 6, and 12 months after treatment to monitor response; with successful treatment, VDRL titers should gradually decrease.
 - (2) Bacterial and viral cultures of lesions
 - (3) Examination of material from base of ulcer
 - (a) Tzanck smear (done with Wright or Giemsa stain) → multinucleated giant cells or immunofluorescent staining (herpes)
 - (b) Darkfield examination → spirochetes (syphilis)
 - (c) Gram stain
 - i. Gram-negative rods in a linear or parallel arrangement (chancroid)
 - ii. "Donovan bodies" (granuloma inguinale)
 - (d) Lymphogranuloma venereum complement fixation test is positive.
- c. Management
 - (1) Herpes
 - (a) Acyclovir
 - i. Primary infections are treated with 200 mg orally 5 × per day (or 400 mg orally 3 × per day) × 7–10 days or until clinical resolution. For proctitis, the dosage should be doubled and continued for a minimum of 10 days.
 - ii. Patients with recurrent episodes may benefit from acyclovir if therapy is started within 24 hours of the appearance of lesions. Dosage regimens are:
 - 400 mg orally every 8 hours × 5 days or
 - 800 mg orally every 12 hours × 5 days
 - iii. Daily suppressive therapy is useful for patients with >6 recurrences per year. The dosage is 400 mg orally bid. Alternative regimens include famciclovir 250 mg orally every 12 hours, valacyclovir 500 mg/day orally, or valacyclovir 1 g/day orally.
 - iv. Patients with severe disease or complications requiring hospitalization are treated with IV acyclovir. The dosage is 5–10 mg/kg IV every 8 hours for 5–7 days or until clinical resolution.
 - (b) Other regimens that are equally efficacious
 - i. Famciclovir 250 mg orally every 8 hours × 7–10 days or
 - ii. Valacyclovir 1 g orally every 12 hours × 7–10 days
 - (c) Symptomatic measures
 - (2) Syphilis
 - (a) Primary, secondary, and early latent (<1 year duration)

- i. Benzathine penicillin G 2.4 million units IM (single dose) *or*
 - ii. Doxycycline 100 mg orally every 12 hours \times 2 weeks only if allergic to penicillin *or*
 - iii. Tetracycline or erythromycin 500 mg orally every 6 hours \times 2 weeks only if allergic to penicillin
- (b) Late latent (>1 year duration) and late syphilis (except neurosyphilis)
 - i. Benzathine penicillin G 2.4 million units IM weekly for 3 doses *or*
 - ii. Doxycycline 100 mg orally every 12 hours \times 4 weeks *or*
 - iii. Tetracycline 500 mg orally every 6 hours \times 4 weeks
- (c) Neurosyphilis
 - i. Aqueous crystalline penicillin G 2–4 million units IV every 4 hours \times 10–14 days *or*
 - ii. Procaine penicillin G 2.4 million units IM every day plus probenecid 500 mg orally every 6 hours for 10–14 days
 - iii. Penicillin-allergic patients require desensitization followed by one of the above regimens. Pregnant patients allergic to penicillin should be desensitized and treated with the appropriate penicillin regimen; all patients with syphilis should be tested for HIV.
- (3) Chancroid (high rate of HIV co-infection)
 - (a) Azithromycin 1 g orally in a single dose *or*
 - (b) Ceftriaxone 250 mg IM in a single dose *or*
 - (c) Erythromycin base 500 mg orally three times a day \times 7 days *or*
 - (d) Ciprofloxacin 500 mg orally every 12 hours \times 3 days
- (4) Granuloma inguinale (all medications must be given for at least 3 weeks and until all lesions have completely healed)
 - (a) Doxycycline 100 mg orally every 12 hours *or*
 - (b) Alternative regimens
 - i. Azithromycin 1 g orally once per week
 - ii. Ciprofloxacin 750 mg orally every 12 hours
 - iii. Erythromycin base 500 mg orally every 6 hours
 - iv. TMP-SMX one double-strength tablet (160 mg/800 mg) orally every 12 hours *or*
- (5) Lymphogranuloma venereum
 - (a) Doxycycline 100 mg orally every 12 hours \times 21 days *or*
 - (b) Erythromycin 500 mg orally every 6 hours \times 21 days
- 6. Fournier gangrene (idiopathic scrotal gangrene)
 - a. Clinical presentation
 - (1) Painful, erythematous or necrotic scrotum, penis, or perineum
 - (2) Febrile and appears toxic
 - (3) Acute onset of pain and swelling
 - (4) Urinary tract symptoms
 - (5) The patient is usually older and immunocompromised in some way (diabetes, chronic steroids, chronic alcoholism).
 - b. *This is a potentially life-threatening disease.*

- c. Etiology
 - (1) Usually due to infection originating from the perianal area
 - (2) Several organisms, both anaerobic and aerobic, are generally involved. *Bacteroides fragilis* is the predominant anaerobe and *E coli* is usually the predominant aerobe. Other agents include hemolytic *Streptococcus*, *Staphylococcus*, and *Clostridia* spp.
- d. Management
 - (1) General supportive measures
 - (2) Cultures
 - (3) Broad-spectrum parenteral antibiotics against anaerobes and gram-negative enteric organisms
 - (4) Prompt urology consult for surgical debridement
 - (5) Hyperbaric oxygen has been used, but its efficacy is unknown and it is rarely necessary.
- 7. Balanoposthitis
 - a. An inflammation of the glans penis (balanitis) and foreskin (posthitis) that is often caused by *Candida*, *Gardnerella*, and anaerobes.
 - b. Recurrent balanoposthitis can be the sole presenting sign of diabetes.
 - c. Clinical presentation
 - (1) Retraction of the foreskin reveals foul, purulent material.
 - (2) The glans is red, swollen, and tender to palpation.
 - d. Management
 - (1) Good hygiene
 - (2) Consider circumcision
 - (3) Antifungal creams (nystatin or clotrimazole)
 - (4) Broad-spectrum antibiotics if secondary infection present (*Streptococcus* infection may be indistinguishable from nonspecific balanitis; cultures or empiric therapy should be considered.)
 - (5) Evaluation for diabetes (if infection recurrent)
 - e. Cases that persist warrant culture and/or biopsy.
- 8. Phimosis
 - a. Definition
 - (1) Inability to retract the foreskin behind the glans
 - (2) Usually secondary to chronic infection of the foreskin associated with progressive scarring
 - b. Management
 - (1) Emergency treatment with a dorsal slit of the foreskin is occasionally required in severe cases.
 - (2) Definitive therapy has traditionally been circumcision (after the infection has been controlled with broad-spectrum antibiotics). Topical steroids (triamcinolone 0.025% every 12 hours or betamethasone 0.05% every day) have been shown to be 70%–90% effective in treating phimosis to allow for some degree of foreskin retraction and avert circumcision.
- 9. Paraphimosis
 - a. Definition: inability to pull retracted foreskin back over the glans

- b. Management
 - (1) Emergency treatment is indicated; vascular compromise can occur.
 - (2) Apply continuous, firm pressure to the glans penis for 5–10 minutes to reduce edema, and then pull the foreskin over the glans.
 - (3) If manual reduction is unsuccessful, infiltrate the constricting ring with a local anesthetic and make a superficial vertical incision dorsally in the midline (dorsal slit).
 - (4) Definitive treatment is circumcision (after inflammation subsides).
- 10. Constriction injuries to the penis
 - a. Must be treated immediately
 - b. Rings, rubber bands, wire, hair, and other objects can transect the urethra and cause neurovascular compromise.
- 11. Fracture of the penis
 - a. Acute tear of the tunica albuginea (usually during erection) causes a swollen (or bent), painful penis.
 - b. Diagnostic evaluation
 - (1) Retrograde urethrogram to exclude associated urethral injury
 - (2) Immediate urologic consult
 - c. Treatment: surgical repair
- 12. Peyronie disease
 - a. Clinical presentation
 - (1) Patients present with a history of dorsal penile curvature with painful erections (and may be associated with Dupuytren contracture of the hand).
 - (2) Examination reveals a thickened plaque involving the tunica albuginea of the corporal bodies.
 - b. Management
 - (1) This is not an emergency.
 - (2) If the patient is bothered by it, urologic referral is in order.
- 13. Carcinoma
 - a. Clinical presentation
 - (1) A nontender or warty growth is usually found on the glans of uncircumcised men >50–60 years old.
 - (2) On physical examination, always retract the foreskin to look for this.
 - b. Management: immediate referral for this aggressive tumor
- 14. Priapism
 - a. Definition: a sustained erection unrelated to sexual stimulation; there are two forms:
 - (1) Low-flow (ischemic) priapism: painful
 - (a) Pathophysiology: venous outflow → venous stasis and ischemia that involves the corpus cavernosae but spares the glans and corpus spongiosum → *rigid, painful* penile shaft and soft glans
 - (b) Because the spongiosum is spared, the patient should be able to void.
 - (c) A surgical emergency: irreversible damage occurs in 24–48 hours.

(d) Etiology

i. Reversible (may respond to medical therapy)

- Sickle cell disease or trait (most common cause in children)
- Intracavernosal injections of papaverine, phentolamine, alprostadil, and prostaglandin E₁ for erectile dysfunction (most common cause in adults)
- Leukemic infiltration

ii. Irreversible (generally unresponsive to medical therapy)

- Medications (antihypertensives, anticoagulants, and antidepressants)
- Illegal substances (alcohol, marijuana, cocaine)
- Idiopathic

(2) High-flow (nonischemic) priapism: rare and usually painless

(a) Not a true emergent condition; long-term sequelae are rare.

(b) Pathophysiology: ↑ arterial blood flow to the corpus cavernosae → ↑ venous blood flow → partially rigid, painless penile shaft and hard glans

(c) Etiology

i. Groin or straddle injury → arterial-cavernosal shunt formation (most common cause)

ii. High spinal cord injury/lesion

b. Management

(1) Obtain immediate urologic consult.

(2) Manage ischemic priapism in a stepwise fashion to achieve resolution as quickly as possible: dry aspiration → aspiration with irrigation → intracavernosus injections of phenylephrine (dilute with normal saline to a concentration of 100–500 mcg/mL)

(a) Repeated injections (1 mL every 3–5 minutes as needed up to 1 hour) should be performed before deciding that this treatment is unsuccessful and a surgical shunt is required.

(b) Monitor for adverse effects during and after intracavernosus injection(s):

- i. Abnormal vital signs (hypertension, tachycardia/bradycardia)
- ii. Cardiac dysrhythmias
- iii. Headache, palpitations

(c) In patients with an underlying disorder (sickle cell disease, hematologic malignancy), systemic treatment of the disorder should be administered concurrently with the intracavernosus injection(s).

(3) Initial management of nonischemic priapism should be observation. Corporal aspiration should be performed only if the diagnosis is in question. Selective arterial embolization is recommended for patients who request treatment.

15. Testicular torsion

a. Incidence may occur at any age but has bimodal peaks: the first few days of life and between ages 12 and 18.

b. Classic clinical scenario

(1) Acute onset of severe, unilateral testicular pain or lower abdominal pain is typical; there may be a recent history of strenuous physical activity or a past history of testicular pain with spontaneous relief.

(2) Swelling occurs within hours; examination (conducted with the patient in the standing position) reveals a swollen, firm, "high-riding" testicle with a transverse

lie; the contralateral testicle may also have a transverse lie, because the underlying anatomic abnormality ("bell-clapper deformity"), which predisposes the patient to torsion, may be bilateral.

- (3) An associated reactive hydrocele may be present; loss of the cremasteric reflex is an associated finding with high specificity, but it is not diagnostic and is not always present.
- (4) Urinary symptoms, pyuria, leukocytosis, and fever are typically absent.
- (5) The most common misdiagnosis is "epididymitis," because there is tenderness posteriorly where the epididymis is normally located. However, what you are actually feeling is a tender testicle, because when the testicle is "torsed" (twisted around), the epididymis is then located anteriorly.

c. Diagnostic evaluation

- (1) Never exclude the diagnosis of testicular torsion based on a single element of the history or physical examination finding.
 - (a) A prior history of torsion and orchiopexy does not exclude the diagnosis (especially if absorbable sutures were used).
 - (b) Up to 80% of patients report anorexia, nausea, and vomiting.
 - (c) Of all the examination findings, presence of the cremasteric reflex is the most helpful in excluding the diagnosis of torsion.
- (2) Ultrasound examination and technetium testicular scanning detect the amount of blood flow to the testicle (with the normal testicle serving as control); flow is decreased or absent on the affected side with torsion. Color Doppler ultrasound is more accurate than the traditional Doppler examination and is now the test of choice in most hospitals. Sensitivity ranges from 83% to 100%; however, sensitivity is decreased in younger age groups, especially neonates.
- (3) *Do not delay urologic consult to wait for a scan.*
- (4) Emergency surgical exploration of the scrotum is the definitive diagnostic test and is indicated if testicular torsion cannot be excluded with certainty.

d. Management

- (1) Obtain immediate urologic consult.
- (2) Order a CBC and urinalysis (usually normal in torsion) and prepare patient for surgery.
- (3) Radionuclide scanning or Doppler examination may be performed if either is immediately available and desired by the urologic consultant, but they should not delay definitive treatment.
- (4) Surgery is indicated as soon as the diagnosis is made, because testicular viability decreases rapidly; salvage rate is 80%–100% up to 6 hours of ischemia, 20% after 10 hours, and approaches zero after 24 hours.
- (5) Symptomatic therapy for patients awaiting surgery is most appropriate.

16. Torsion of appendices epididymis and testis

- a. These appendages are small pedunculated structures (without any known function) attached to the epididymis and testis that can torse and produce pain.
- b. Clinical presentation
 - (1) Early on, pain is localized and a tender nodule is palpated. The "blue dot sign" on transillumination of the testis is pathognomonic.
 - (2) If seen late, pain is diffuse and swelling is present, making diagnosis more difficult.

c. Management

- (1) If the diagnosis is certain, surgery is not required because these structures calcify.
- (2) If the diagnosis is uncertain, surgery is indicated to exclude testicular torsion.

17. Epididymitis

a. Clinical presentation

- (1) Inflammation of the epididymis is usually caused by bacterial infection and is more common in young adults than in preadolescent boys or older men.
- (2) There is a gradual onset of unilateral pain and swelling (sudden onset is more common with torsion).
- (3) Associated fever and dysuria are common.
- (4) On examination, the epididymis is tender, swollen, and located in its normal posterior position.
- (5) Elevation of the testicle is associated with relief of pain (Prehn sign), but this sign is unreliable; it cannot be used to distinguish epididymitis from torsion.

b. Etiology is age dependent.

- (1) *Chlamydia trachomatis* (usually sexually transmitted) is the most common pathogen (followed by *Neisseria gonorrhoeae*) in patients <35 years old.
- (2) *E coli* and *Pseudomonas* (not sexually transmitted) are the most common pathogens in patients >35 years old.
- (3) Fungal infection must be considered in homosexual men with epididymitis or epididymo-orchitis.

c. Diagnostic evaluation

- (1) Urinalysis: a few WBCs are common (but may also be seen with torsion).
- (2) Urine culture
- (3) CBC: a leukocytosis of 10,000–30,000/mm³ is common.
- (4) Urethral Gram stain
- (5) Urethral culture for gonorrhea and chlamydia

d. Management

(1) Supportive measures

- (a) Bed rest initially, followed by scrotal support
- (b) Ice packs
- (c) Analgesics
- (d) Fecal softeners

(2) Start antibiotics: antibiotic selection should be based on the patient's age and presumed pathogens.

(a) Homosexual or sexually active men (coliforms)

- i. Ciprofloxacin 500 mg orally every 12 hours or ofloxacin 200 mg orally every 12 hours × 10–14 days is the usual regimen.
- ii. If <17 years old: amoxicillin-clavulanate or TMP-SMX orally

(b) Heterosexual men <35 years old (gonorrhea, Chlamydia, and possibly coliforms)

- i. Ceftriaxone 250 mg IM and doxycycline 100 mg orally every 12 hours × 10 days or ofloxacin 300 mg orally every 12 hours × 10 days

- ii. Cautionary advice
 - Ciprofloxacin is ineffective against *Chlamydia*.
 - Single-dose therapy (while appropriate for simple urethritis) is inadequate for simple epididymitis.
 - (c) Heterosexual men >35 years old or bisexual men (usually coliforms, but gonorrhea and chlamydia may contribute): levofloxacin 500 mg/day orally or ofloxacin 300 mg orally bid × 10 days
 - (3) Refer to urologist for follow-up in 5–7 days.
 - (4) Admission for IV antibiotics is indicated for:
 - (a) Toxic-appearing patients (particularly those who are older and in whom a scrotal abscess is suspected)
 - (b) Immunosuppressed patients
 - (c) Patients with severe bilateral epididymitis
18. Urethral stricture
- a. Etiology: trauma, urethral instrumentation, or a complication of chlamydial or gonorrheal infection
 - b. Diagnostic evaluation
 - (1) History of partial or complete retention
 - (2) If unable to pass a catheter, a retrograde urethrogram can determine the extent and location of the stricture.
 - c. Management
 - (1) Catheterization
 - (2) If unable to pass a regular or Coudé catheter after 2 or 3 careful attempts, obtain a urologic consult for catheterization using filiforms.
19. Urethral foreign bodies
- a. Occur at any age
 - b. Clinical presentation: hematuria or signs and symptoms of obstruction or infection
 - c. Diagnosis: made by palpation and confirmed on plain radiograph, retrograde urethrogram, or cystoscopy
 - d. Management: urologic consult for removal
20. Urinary retention
- a. Etiology
 - (1) Benign prostatic hypertrophy with bladder neck obstruction (most common cause in men >50 years old)
 - (2) Strictures (history of a sexually transmitted infection or pelvic trauma)
 - (3) Drugs
 - (a) Antihistamines
 - (b) Anticholinergic agents
 - (c) Antispasmodic agents
 - (d) Cyclic antidepressants
 - (e) α -adrenergic stimulants
 - (f) Antipsychotic agents
 - (4) Meatal stenosis
 - (5) Bladder neck contracture

- (6) Bladder cancer
- (7) Cancer of the prostate
- (8) Neurogenic
- b. Clinical presentation
 - (1) The patient is in distress (unless he has a neurogenic bladder) and complains of hesitancy or poor stream followed by low abdominal pain and inability to void for 6–8 hours.
 - (2) On physical examination, the bladder is often visibly distended and easily palpable.
- c. Diagnostic evaluation
 - (1) Urinalysis to exclude co-infection
 - (2) BUN/creatinine to assess renal function
- d. Management
 - (1) Pass a Foley catheter; this is both diagnostic and therapeutic.
 - (2) Observe patient with chronic urinary retention for 2–4 hours after relief of urinary retention for development of postobstructive diuresis.
 - (a) Syndrome of massive urine output that can produce volume depletion, electrolyte imbalance, and hypotension
 - (b) These patients require hospitalization and additional laboratory studies (serum and urine electrolytes).
 - (3) Most other patients can be discharged to home with the catheter in place and referred to a urologist for evaluation.
 - (4) Antibiotics (trimethoprim or TMP-SMX) should be prescribed if a concomitant urinary tract infection is present.
 - (5) Belladonna and opium suppositories should not be prescribed for these patients. Continued use will prevent a successful voiding trial when the catheter is removed.

RENAL AND UROLOGIC DISORDERS: PRACTICE CLINICAL SCENARIOS

Answers immediately follow the practice clinical scenarios.

Scenario A

Presentation: A 7-year-old child presents with facial swelling and "dark urine." The child had a sore throat last week.

What is the diagnosis?

Scenario B

Presentation: A 60-year-old man on peritoneal dialysis presents with fever and abdominal pain. He states the dialysate fluid on return is "cloudy."

What is the diagnosis?

Scenario C

Presentation: A 57-year-old diabetic man has pain in his scrotum.

Physical examination: There is evidence of ecchymosis with surrounding erythema.

What is the diagnosis?

Scenario D

Presentation: An 15-year-old male has pain on the right side of his flank and testicle.

Physical examination: The testicle is swollen, firm, and high-riding with a transverse lie; the cremasteric reflex is absent.

What is the diagnosis?

Scenario E

Presentation: A pregnant woman presents with urinary tract symptoms of dysuria. Urinalysis shows 50 WBC/high-power field on a centrifuged specimen.

What is the diagnosis?

ANSWERS TO PRACTICE CLINICAL SCENARIOS

Scenario A

Diagnosis: acute glomerulonephritis

Diagnostic evaluation: Urine sediment shows RBCs, RBC casts, and proteinuria.

Scenario B

Diagnosis: spontaneous bacterial peritonitis

Scenario C

Diagnosis: Fournier gangrene

Management: Management includes general supportive measures, cultures, and broad-spectrum parenteral antibiotics against anaerobes and gram-negative enteric organisms. Prompt urology consult should be obtained for surgical debridement. Hyperbaric oxygen has been used, but its efficacy is unknown and it is rarely necessary.

Scenario D

Diagnosis: testicular torsion

Diagnostic evaluation: Never exclude the diagnosis of testicular torsion based on a single element of the history or physical examination finding. A prior history of torsion and orchiopexy does not exclude the diagnosis (especially if absorbable sutures were used). Up to 80% of patients report anorexia, nausea, and vomiting. Of all the examination findings, presence of the cremasteric reflex is the most helpful in excluding the diagnosis of torsion.

Ultrasound examination and technetium testicular scanning detect the amount of blood flow to the testicle (with the normal testicle serving as control); flow is decreased or absent on the affected side with torsion. Color Doppler ultrasound is more accurate than the traditional Doppler examination and is now the test of choice in most hospitals. Sensitivity ranges from 83% to 100%; however, sensitivity is decreased in younger age groups, especially neonates. *Do not delay urologic consult to wait for a scan.*

Emergency surgical exploration of the scrotum is the definitive diagnostic test and is indicated if testicular torsion cannot be excluded with certainty.

Scenario E**Diagnosis:** UTI

Management: In pregnant patients, ampicillin, amoxicillin, and cephalosporins remain first-line agents despite increasing resistance rates. A course of 10–14 days is recommended. Nitrofurantoin is becoming a first-line agent because it is efficacious, inexpensive, and well-tolerated; because of its short half-life, it must be taken for at least 7 days. Do not give to patients with glucose-6-phosphate dehydrogenase deficiency.

NOTES

CUTANEOUS DISORDERS

Introduction.....	1055
General Approach to the Patient Presenting with a Rash	1055
Toxicodendrons (Poison Ivy, Poison Oak, Poison Sumac)	1056
Diaper Dermatitis	1057
Exfoliative Dermatitis/Exfoliative Erythroderma Syndrome.....	1058
Erythema Multiforme	1059
Impetigo.....	1064
Cellulitis.....	1066
Cutaneous Abscess.....	1068
Staphylococcal Scalded Skin Syndrome	1070
Pemphigus Vulgaris	1071
Herpes Infections.....	1071
Herpes Simplex	1071
Herpes Zoster (Shingles)	1073
Fungal Disorders.....	1075
Erythema Nodosum.....	1078
Decubitus Ulcer.....	1079

CUTANEOUS DISORDERS: SELF-ASSESSMENT QUESTIONS

1. Precipitants of erythema multiforme include all of the following except:
 - (a) Drugs
 - (b) Infectious diseases
 - (c) Malignancies (lymphoma, carcinoma)
 - (d) Trauma
2. The presence of "target lesions" (erythematous plaques with dusky centers and bright red borders) on the palms and soles is classically associated with:
 - (a) Erythema multiforme
 - (b) Secondary syphilis
 - (c) Disseminated gonococcal disease
 - (d) Pityriasis rosea
3. A positive Nikolsky sign is typical of skin lesions associated with:
 - (a) Toxic shock syndrome
 - (b) Herpes zoster
 - (c) Toxic epidermal necrolysis
 - (d) Contact dermatitis
4. A Tzanck smear can be useful in making the diagnosis of all of the following except:
 - (a) Herpes simplex (types 1 and 2)
 - (b) Varicella (chickenpox)
 - (c) Erythema multiforme
 - (d) Herpes zoster (shingles)
5. All of the following statements regarding erysipelas are true except:
 - (a) It is seen most commonly on the face and the lower extremities.
 - (b) It is caused by *Staphylococcus aureus*.
 - (c) It is a type of cellulitis.
 - (d) Examination typically reveals a tender, red plaque with raised and sharply demarcated borders.
6. A pink to red maculopapular rash that starts on the face and rapidly spreads to the trunk and extremities and is associated with lymphadenopathy (especially postauricular, suboccipital, and posterior cervical) is most characteristic of:
 - (a) Rubella
 - (b) Roseola
 - (c) Rubella
 - (d) Varicella

7. All of the following statements about "Koplik spots" are true except:
 - (a) They are found on the buccal mucosa.
 - (b) They are pathognomonic of rubella.
 - (c) They are described as white papules on an erythematous base.
 - (d) They appear 1–2 days before the onset of the rash associated with measles.
8. A herald patch is most closely associated with:
 - (a) Psoriasis
 - (b) Exfoliative dermatitis
 - (c) Toxic shock syndrome
 - (d) Pityriasis rosea
9. Mucous membrane involvement may be seen in all of the following disorders except:
 - (a) Stevens-Johnson syndrome
 - (b) Erythema multiforme
 - (c) Toxic epidermal necrolysis
 - (d) Staphylococcal scalded skin syndrome
10. Which of the following statements regarding oral acyclovir in the treatment of herpes simplex is true?
 - (a) It is recommended for the treatment of primary infections, especially urogenital infections.
 - (b) It decreases viral shedding, accelerates healing, and shortens the duration of symptoms.
 - (c) It may be used prophylactically in patients who have very frequent recurrences.
 - (d) All of the above
11. All of the following factors are useful in distinguishing toxic epidermal necrolysis from staphylococcal-induced scalded skin syndrome except:
 - (a) Presence of a positive Nikolsky sign
 - (b) Age group affected
 - (c) Presence of mucous membrane involvement
 - (d) History of recent upper respiratory infection or purulent conjunctivitis
12. All of the following conditions classically produce lesions involving the soles and palms except:
 - (a) Erythema multiforme
 - (b) Chickenpox
 - (c) Secondary syphilis
 - (d) Hand-foot-and-mouth disease

13. An immunocompromised patient presents with a painful vesicular rash that has a dermatomal distribution. The most appropriate management for this patient is:
- (a) Oral acyclovir
 - (b) Analgesics and drying compresses
 - (c) Systemic corticosteroids
 - (d) IV acyclovir and hospital admission
14. All of the following statements are true of disseminated gonococcal disease except:
- (a) It is more common in men than women.
 - (b) The lesions begin as tender red papules or petechiae and evolve into pustules and vesicles with a "red halo" and a gray necrotic center.
 - (c) The lesions are generally distributed over the periarticular regions of the distal extremities.
 - (d) Associated fever, chills, and migratory polyarthralgias are common.
15. Complications of impetigo contagiosa include all of the following except:
- (a) Local spread (autoinoculation)
 - (b) Spread to others
 - (c) Acute poststreptococcal glomerulonephritis
 - (d) Rheumatic fever
16. The following statements regarding herpes zoster are accurate except:
- (a) It is a communicable disease.
 - (b) Lesions at the tip of the nose indicate possible eye involvement.
 - (c) The cervical dermatome is the most commonly involved.
 - (d) Post-herpetic neuralgia, a complication of this infection, is seen in 40% of patients >60 years old.
17. The lesions of erythema nodosum are most commonly found on the:
- (a) Arms
 - (b) Face
 - (c) Legs
 - (d) Trunk

ANSWERS

- | | | | | | |
|------|------|------|-------|-------|-------|
| 1. d | 4. c | 7. b | 10. d | 13. d | 16. c |
| 2. a | 5. b | 8. d | 11. a | 14. a | 17. c |
| 3. c | 6. a | 9. d | 12. b | 15. d | |

Use the pre-chapter multiple choice question worksheet (page xx) to record and determine the percentage of correct answers for this chapter.

I. INTRODUCTION

Cutaneous disorders comprise a small portion (1%) of the examination content. Pictorial identification is especially important. The lesions and rashes you are likely to be asked to identify are noted in this chapter. You may also wish to consult a color dermatology atlas, such as Fitzpatrick's Color Atlas and Synopsis of Clinical Dermatology, Weston and Lane's Color Textbook of Pediatric Dermatology, and/or Knoop's Atlas of Emergency Medicine. You should be familiar with all the color plates in the Seventh Edition of Tintinalli's text.

II. GENERAL APPROACH TO THE PATIENT PRESENTING WITH A RASH

- A. Inquire about prodromal symptoms and antecedent events (eg, new medications).
- B. Note patient's age, immune status, past medical history and medications, allergies, and presence/absence of toxicity.
- C. Examine the rash and determine other characteristics.
 1. Note character of rash and other cutaneous lesions.
 - a. Macular → flat and ≤ 1 cm
 - b. Patchy → flat and > 1 cm
 - c. Papular → raised and ≤ 1 cm
 - d. Plaque → raised and > 1 cm
 - e. Maculopapular, nodular → dermal or subcutaneous solid lesion 1–2 cm
 - f. Tumor → dermal or subcutaneous solid lesion > 2 cm
 - g. Vesicular → blister ≤ 1 cm
 - h. Bullous → blister > 1 cm
 - i. Pustules → small blister containing purulent material
 - j. Scales or keratoses → built up epidermis
 - k. Crusts, erosions → loss of part or all of epidermis
 - l. Ulceration → loss of dermis or deeper
 2. Determine where it started and how it evolved.
 3. Note overall distribution, pattern, and configuration, including involvement of mucous membranes and/or palms and soles.
 4. Determine if pruritic or painful.
 5. Determine what, if anything, the patient has already done to treat the rash (eg, applied hydrocortisone cream or a neomycin-containing antibacterial ointment), because this may have changed the appearance of the rash or caused a secondary contact dermatitis.

III. TOXICODENDRONS (POISON IVY, POISON OAK, POISON SUMAC)

A. Overview

1. Toxicodendrons are responsible for more cases of allergic contact dermatitis in the United States than all other allergens combined.
2. The development of a reaction requires prior sensitization (days or years earlier); these are type IV cell-mediated, delayed hypersensitivity reactions.
3. The antigen responsible for producing the skin reaction that occurs is urushiol, which is found in poison ivy, poison oak, poison sumac, and mangoes.

B. Clinical presentation

1. The rash is characterized by erythema (frequently in a linear configuration from brushing against a branch) and itching. It may also be associated with papules, vesicles, or bullae.



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2. The rash usually appears 5–21 days after a first-time exposure; for subsequent exposures, onset begins any time from 6 hours to 3 days, depending on the severity of the exposure.
 - a. Mild exposure → onset in 2–3 days and lasts 1–2 weeks
 - b. Severe exposure → onset in 6–12 hours and lasts 2–3 weeks
3. A widely held misconception is that rupture of the vesicles spreads the rash, but the blister fluid does not contain antigen and therefore cannot cause spread of the rash. Of note, different areas of the skin can react at different times, furthering this misconception.

C. Management

1. Mild dermatitis: calamine lotion, oatmeal colloidal bath, a topical steroid preparation, and an oral antihistamine
2. Moderate to severe dermatitis
 - a. Wet-to-dry compresses with water or aluminum acetate, eg, Burow solution
 - b. Oral antihistamines (eg, hydroxyzine, diphenhydramine)
 - c. Systemic corticosteroids are indicated for severe reactions. They should be continued for 2–3 weeks, with a gradual taper to avoid rebound. A typical regimen recommended by dermatologists is prednisone 60 mg/day × 5 days, then 40 mg/day × 5 days, then 20 mg/day × 5 days (can be extended to 21 days for more severe conditions).

D. Prophylaxis

1. If reexposure occurs, the patient should immediately wash the area with cold soapy water. Urushiol is absorbed in 10–20 minutes, so the allergen must be removed quickly.
2. This may prevent the dermatitis from developing, or at least lessen its severity.

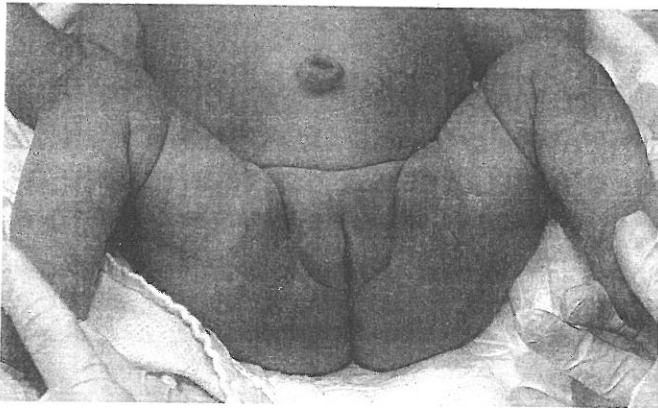
IV. DIAPER DERMATITIS

A. Overview

1. This is not a specific entity but rather a nonspecific term referring to several dermatologic conditions that occur in the diaper area. Be able to distinguish irritant, contact dermatitis, and *Candida* diaper rashes.
2. The most important inciting factor is constant moisture. Other factors are diaper detergents, disinfectants, an alkaline pH, fecal material, and intestinal enzymes.
3. The effects of local irritants are amplified by infrequent diaper changes, inadequate skin cleansing, and occlusive diapers.
4. Incidence is highest in babies 9–12 months old. It can also be seen in incontinent and paralyzed adults and may be a red flag for neglect or abuse in this population.

B. Clinical presentation

1. The rash begins as an erythematous eruption over areas of increased friction of skin against the diaper (buttocks, genitals, lower abdomen, and thighs). This progresses to papules, vesicles, erosions, and ulcers.



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2. It may reflect one or a combination of the following:
 - a. An underlying skin condition such as atopic or seborrheic dermatitis: a clue is the presence of concurrent facial lesions (atopic dermatitis) or scalp lesions (seborrheic dermatitis).
 - b. Primary irritant contact dermatitis: may be from ammonia and bacterially produced putrefactive enzymes (the odors are characteristic and the skin folds are notably spared), or from a new soap, shampoo, laundry detergent, etc.
 - c. Secondary infection with *Candida albicans*: a clue is the classic appearance of the rash (moist, beefy-red plaques with wellmarked edges in association with satellite lesions) and a duration >3 days.

C. Management

1. General measures
 - a. Discontinue use of plastic or rubber occlusive diaper pants.
 - b. Use supra-absorbent disposable diapers or cloth diapers, and change them frequently.
 - c. Avoid the use of harsh cleansing agents, even "baby wipes" and soaps (which remove protective skin oils); use tepid water, and pat dry.
 - d. Leave diapers off for extended periods of time or dry diaper area with hairdryer.
 - e. Protect the skin from maceration with a barrier cream such as commercial zinc oxide diaper rash creams or A+D® ointment. Do not use baby powder with cornstarch, which is metabolized by bacteria.
2. Additional specific measures
 - a. For mild inflammation secondary to atopic, seborrheic, or contact dermatitis, use 1% hydrocortisone cream with a barrier, such as zinc oxide ointment or petrolatum gel (to prevent it from washing off).
 - b. If the rash is more severe, persists >3–4 days, or there are overt signs of *Candida* infection, add nystatin, clotrimazole, or miconazole cream.

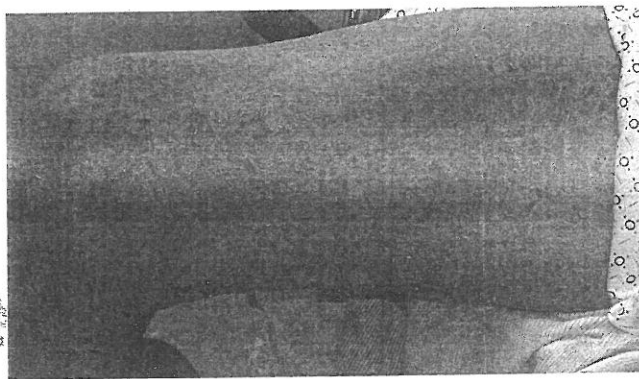
V. EXFOLIATIVE DERMATITIS/EXFOLIATIVE ERYTHRODERMA SYNDROME

A. Etiology

1. Idiopathic most common
2. Drug-induced (>50 drugs have been implicated)
3. Underlying malignancy (lymphoma, leukemia, or other lymphoreticular malignancy) or immunosuppression (HIV)
4. Preexisting dermatoses most common (psoriasis, eczema, seborrhea, etc)
5. Allergic contact dermatitis

B. Clinical presentation

1. Classic clinical scenario: A 42-year-old man with a past medical history of seizures on a new medication appears ill and complains of itching, a chilly sensation, and "tightness" of the skin. He has a low-grade fever and is hypotensive and tachycardic. On examination, a scaly, warm, erythematous rash is covering >50% of his body surface area. There is no oral involvement, and the rash is not tender to the touch. Nikolsky sign is negative.



Courtesy of David Effron, MD, FACEP

2. Other findings may include fever or hypothermia, dehydration, lymphadenopathy, hepatosplenomegaly, alopecia, or gynecomastia.
3. Because of increased blood flow to the skin, the patient may have high output heart failure.

C. Diagnostic evaluation

1. Consider CBC, serum chemistries, liver function tests, erythrocyte sedimentation rate, and urinalysis. Consider other tests (HIV, peripheral blood smear) as indicated.
2. Because this disorder is usually the result of either an underlying cutaneous or systemic disease or a response to a drug or chemical, patients should be admitted for a diagnostic evaluation. Mortality is as high as 30%.
3. Skin biopsy (lymph node biopsy if significant lymphadenopathy)

D. Differential diagnosis

1. Erythema multiforme
2. Toxic epidermal necrolysis
3. Toxic shock syndrome
4. Staphylococcal scalded skin syndrome
5. Kawasaki disease (children)

E. Management

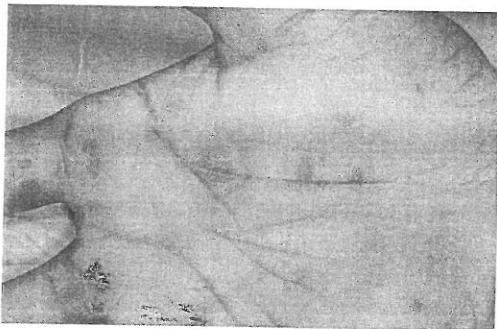
1. Goal is to correct/eliminate the underlying cause while providing symptomatic relief and maintaining skin moisture.
2. Antihistamines and topical steroids are effective in controlling the symptoms in most instances and may need to be continued for weeks or months.
3. Water baths with bath oils and skin emollients are also helpful.
4. Severe or resistant cases are treated with systemic corticosteroids.

VI. ERYTHEMA MULTIFORME (EM)

A. Overview

There has been controversy regarding the nomenclature of EM through the years, but there are three generally recognized subsets: erythema multiforme minor, erythema multiforme major (or bullous EM), and the spectrum of Stevens-Johnson syndrome (SJS) → SJS/toxic epidermal necrolysis (TEN) overlap → TEN

1. EM minor: target lesions or raised papules distributed acrally; no mucous membrane involvement



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2. EM major (or bullous EM): <10% body surface area (BSA) blistering and epidermal detachment plus typical target lesions or raised atypical target lesions; possible mucous membrane involvement
3. SJS: <10% BSA blistering and epidermal detachment plus widespread erythematous or purpuric macules or flat atypical target lesions; primarily on the trunk and face; mucous membrane involvement
4. Overlap SJS/TEN: 10%–30% BSA blistering and epidermal detachment plus widespread purpuric macules or flat atypical target lesions; mucous membrane involvement
5. TEN: ≥30% BSA blistering and epidermal detachment with widespread purpuric macules or flat atypical target lesions; mucous membrane involvement

Table 45: Classification of Erythema Multiforme

	EM Minor	EM Major	SJS* — SJS/TEN — TEN
Distribution	Extremities	Extremities	Widespread
Population	Children and young adult men	Young adult men (60%)	Middle-aged women (50%–60%)
Mucous membrane involvement	No	Sometimes	Yes
Recurrent	Yes	Yes	Sometimes
Associated with	Viral infection	Infection (particularly herpes) and drugs	Mostly drugs (75%–95%), also HIV, collagen vascular disease and cancer
Clinical course	Benign	Benign; death rare	Death rate: SJS 3%, SJS-TEN 6%, TEN 39%

*Associated with *Mycoplasma pneumonia* in children

B. Etiology (none found in 50% of cases)

1. Infections (predominant in children); examples include:
 - a. Herpes simplex types I and II (75% of all children experience recurrence of erythema multiforme)
 - b. Influenza type A
 - c. Hepatitis
 - d. *Mycoplasma pneumonia*
 - e. Mumps
 - f. Histoplasmosis
 - g. Streptococci
2. Drugs, collagen vascular disorders, and malignancies (more common causes in adults)
 - a. Commonly implicated drugs
 - (1) Antibiotics (sulfonamides, penicillins, cephalosporins)
 - (2) Anticonvulsants (phenytoin, carbamazepine, lamotrigine)
 - (3) Allopurinol
 - (4) Sulfonyleureas
 - (5) NSAIDs
 - (6) Barbiturates

- b. Connective tissue disorders
 - (1) Systemic lupus erythematosus
 - (2) Rheumatoid arthritis
 - (3) Periarthritis nodosa
 - (4) Dermatomyositis
- c. Malignancies
 - (1) Leukemia
 - (2) Lymphoma
 - (3) Carcinoma

C. EM major

1. Lesions are "multiform" and usually symmetric in distribution (back of the hands or feet, extensor surfaces of the extremities); they also occur on the face, palms, and soles and may be generalized.
2. The characteristic lesions are erythematous macules with a dusky, violaceous, or bullous center found on the palms and soles. Prodromal symptoms are conspicuously absent.
3. Mucosal involvement (when it occurs) is usually oral but may be ocular.
4. The lesions usually start as erythematous macules or papules that may enlarge or coalesce to form urticarial-like plaques.
5. Vesiculobullous lesions develop centrally within preexisting macules, papules, or urticarial wheals.
6. The classic "iris" or "target" lesions are erythematous plaques with dusky centers and bright red borders resembling a bull's-eye and are most commonly found on the hands or wrists.



Courtesy of David Effron, MD, FACEP

D. SJS

1. Most cases are related to drug ingestion (anticonvulsants, sulfas, and NSAIDs); potential for fatal outcomes
2. Characterized by widespread bullous lesions, severe mucous membrane involvement, and multisystem pathology



Courtesy of David Effron, MD, FACEP

3. **Classic clinical scenario:** The patient appears very ill and gives a history of fever, malaise, myalgias, and arthralgias. This is followed by abrupt onset of bullous mucocutaneous lesions that subsequently erode. Multiple mucosal surfaces (eyes, mouth, lips, urogenital area, and anus) are involved. Patients often cannot eat because of painful stomatitis. Conjunctivitis is common, and vesicles on the conjunctiva are sometimes seen; the eyelids may be red, swollen, and crusted. An overall skin examination is likely to reveal widespread erythematous (or purpuric) macules or flat atypical "targets."
4. **Associated with significant morbidity and mortality**
 - a. Denuded skin and mucous membranes result in fluid loss and are susceptible to secondary bacterial infection.
 - b. Significant ocular sequelae (corneal ulceration, blindness) are common.
 - c. Renal involvement (hematuria, renal tubular necrosis, renal failure) can occur but is rare.
 - d. Death, when it occurs, is most often due to fulminant sepsis.

E. Management of EM and SJS

1. Consult with a dermatologist.
2. Identify the precipitant cause (if possible) and treat accordingly. Obtain a medication history, and discontinue any suspicious drug (in consultation with the physician who prescribed it). If the patient has a herpes simplex infection, antiviral therapy is indicated.
3. Patients with EM minor can be discharged on topical steroids (not to be applied to eroded areas) and symptomatic treatment for pruritus, oral lesions, bullous/erosive lesions, nausea, etc (Burrow's solution compresses followed by silvadene application for cutaneous lesions).
4. Hospitalization, IV fluid hydration, and steroids are indicated with significant mucous membrane involvement. Systemic steroids provide symptomatic relief and may reduce morbidity and improve outcome if administered promptly; however, there is no hard evidence that they are of benefit.
5. Obtain ophthalmology consult for patients with ocular lesions.

F. TEN**1. Etiology**

- a. Drugs are the most common cause; the agents most often implicated are the sulfonamides, aromatic anticonvulsants (carbamazepine, phenobarbital, phenytoin), aminopenicillins (ampicillin, amoxicillin), NSAIDs, cephalosporins, and allopurinol.
- b. Other causes include immunizations, chemical agents, infections (especially *Mycoplasma*), connective tissue disorders, leukemia, and lymphoma.

2. Clinical presentation

- a. Primarily a disease of adults (particularly the elderly)
- b. Typically characterized by tenderness and erythema of the skin and mucosa, followed by extensive cutaneous and mucosal blistering as well as desquamation



Courtesy of David Effron, MD, FACEP

- c. A flu-like prodrome often precedes development of the mucocutaneous lesions by 1–14 days.
 - d. The lesions usually begin on the face or upper body as macules, targets, and atypical target lesions before they rapidly spread. The involved epidermis (10%–30% BSA) becomes necrotic and sloughs in large sheets, leaving behind exposed dermis.
 - e. Mucous membrane involvement is the rule; conjunctival involvement is particularly common and associated with significant long-term morbidity from scarring.
 - f. Nikolsky sign is positive.
 - g. The skin cleavage plane is at the dermoepidermal junction; therefore, the entire epidermis is shed.
 - h. Mortality rate is high (almost 40%) and due to massive fluid loss and secondary infections.
- 3. Management is similar to that of second-degree burns.**
- a. Admission, usually to a burn unit
 - b. IV fluid resuscitation to correct hypovolemia and electrolyte abnormalities
 - c. Cessation of suspected drug(s)
 - d. Dermatology consult
 - e. Ophthalmology consult if there is ocular involvement

- f. Close monitoring for infections (avoid prophylactic antibiotics)
- g. Systemic steroids are not recommended; to date, no study demonstrates their efficacy in TEN and they can increase the risk of infection, especially in advanced cases.

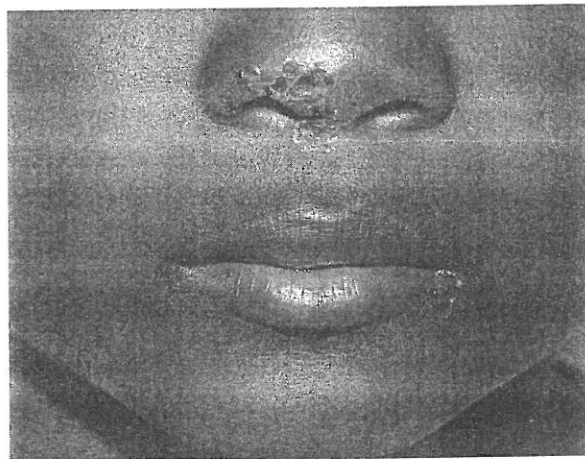
VII. IMPETIGO

A. Overview

1. Most common skin infection seen in the emergency department
2. Superficial gram-positive bacterial skin infection that can occur in any group but is most prevalent in children <6 years old
3. Predisposing factors include warm temperatures, tropical climates, overcrowding, poor hygiene, and an interruption of the natural skin barrier by cuts, abrasions, insect bites, or dermatoses.
4. Highly communicable, both to other body sites (autoinoculation) and other people, particularly in children
5. Lesions may be pruritic but typically are not painful.

B. Two clinically distinguishable variants: impetigo contagiosa and bullous impetigo

1. Impetigo contagiosa (most common variant)
 - a. Etiology: caused by *Staphylococcus aureus* or group A β -hemolytic streptococci (including nephrogenic strains); *S aureus* is currently the most common pathogen.
 - b. Distribution: lesions most commonly seen on the face (around the nose and mouth) and extremities.

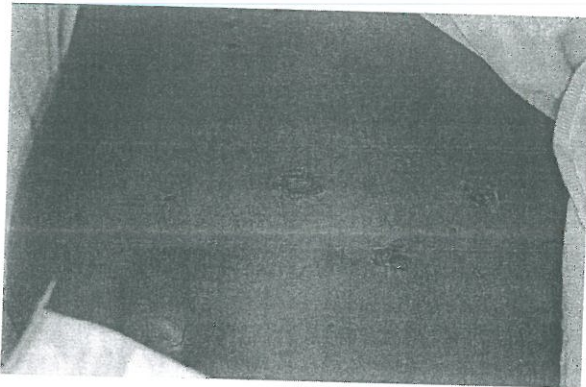


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C. Lesions

- (1) The lesions begin as small pustules or vesicles with erythematous margins. These lesions rupture, leaving behind superficial erythematous erosions with thick, honey-colored crusts.
- (2) Some surrounding erythema may be present when a group A β -hemolytic streptococci is the causative agent.
- (3) Associated regional lymphadenopathy is common.

2. Bullous impetigo is seen most often in neonates.
 - a. Etiology: caused by *S aureus* of phage group 2
 - b. Distribution: lesions most commonly seen in the periumbilical region (or the perineum) in neonates and on the extremities in older children



Courtesy of David Effron, MD, FACEP

- c. Lesions
 - (1) The lesions are flaccid, thin-walled bullae (1–3 cm in diameter) that contain a purulent material. The bullae rupture, leaving shiny, rounded, erythematous erosions with peeling edges ("coin" lesions).
 - (2) There is no surrounding erythema.
 - (3) Associated regional lymphadenopathy is rare.
 - (4) Nikolsky sign is negative.
3. Ecthyma is a condition very close to impetigo that is usually caused by streptococci. The lesions are typically on the legs and begin as pustules that erode and then ulcerate. The infection is usually deeper than impetigo, painful, and has a high rate of scarring.

C. Management

1. Antibiotics (oral or topical)
 - a. Usually treated with a 10-day course of an oral antibiotic effective against both staphylococci and streptococci
 - (1) β -lactamase-resistant antibiotics are first-line treatment (dicloxacillin, cephalosporins, amoxicillin-clavulanate).
 - (2) Clindamycin or TMP-SMX for suspected methicillin-resistant *Staphylococcus aureus* (MRSA)
 - (3) Clindamycin or azithromycin if penicillin-allergic
 - b. Topical antibiotic that can be used alone for limited infections
 - (1) Mupirocin 2% is the classic choice.
 - (2) Retapamulin ointment is a new class of topical antibiotics approved for treatment of impetigo.
2. Good hand washing and personal hygiene limit spread of the infection to others.

D. Complications (focal and systemic)

1. Spread (both locally and to others); children should not return to school until after 24 hours of antibiotic treatment.
2. Cellulitis

3. Regional lymphadenitis
4. Staphylococcal scalded skin syndrome
5. Acute poststreptococcal glomerulonephritis (incidence <1%)
 - a. A nonsuppurative complication in patients infected with nephrogenic strains of streptococci
 - b. Not prevented by antibiotic therapy
 - c. Almost all patients recover, even those who develop renal failure.
6. Rheumatic fever is *not* a sequela of streptococcal skin infections.

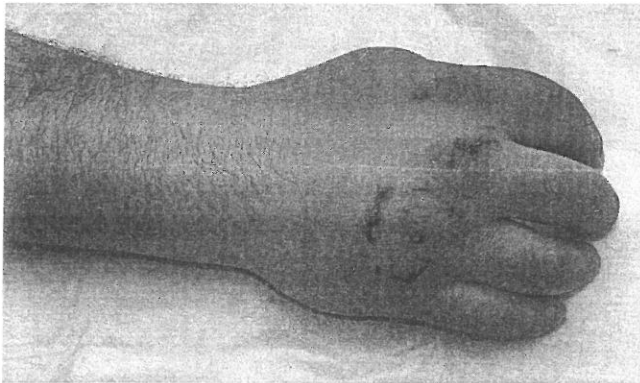
VIII. CELLULITIS

A. Definition and epidemiology

1. Cellulitis is a local soft-tissue inflammatory reaction secondary to bacterial invasion of the skin.
2. While cellulitis is classically associated with comorbidities such as diabetes or peripheral vascular disease, <5% of all patients have such coexisting conditions.
3. Most infections involve the extremities.

B. Clinical presentation

1. The classic symptoms are the result of a localized inflammatory reaction characterized by pain, warmth, erythema, and induration. This reaction is a result of bacterial invasion that is most often strains of staphylococci or streptococci.

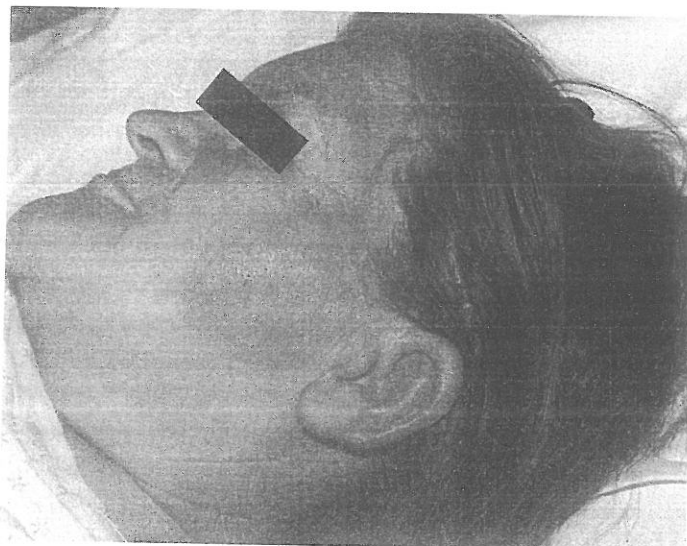


Courtesy of David Effron, MD, FACEP

2. Systemic involvement such as fever and bacteremia can occur and is most common in immunosuppressed patients.

C. Specific types

1. Erysipelas
 - a. Etiology: group A streptococci, usually *Streptococcus pyogenes*
 - b. Epidemiology: generally affects patients >60 years old and is associated with prodrome of fever, chills, and malaise
 - c. Clinical presentation
 - (1) Acute cellulitis characterized by a bright red color, sharp margins, and severe pain



Courtesy of David Effron, MD, FACEP

- (2) Involves upper dermis, lymphatics, and superficial subcutaneous tissue
- 2. Periorbital cellulitis (see page 152)
- 3. Necrotizing cellulitis
 - a. Etiology
 - (1) Clostridial species are the most common pathogens.
 - (2) Clostridial infection is typically associated with significant gas production in the subcutaneous tissue.
 - b. Epidemiology
 - (1) A superficial necrotizing soft-tissue infection limited to subcutaneous tissue; characterized by rapid progression, skin sloughing, and gas in the tissue
 - (2) Most often associated with preceding trauma or surgery
- 4. Community-associated methicillin-resistant *Staphylococcus aureus* (MRSA)
 - a. Emerged as prevalent pathogen for many types of soft-tissue infections
 - b. White race and the presence of furuncles (boils) appear to be the strongest predictors of community-associated MRSA colonization and infection.
 - c. Other strong predictors of colonization include a history of recurrent abscess and homelessness.

D. Diagnostic evaluation

- 1. Several specific causes and types of cellulitis must be considered; however, the ability to isolate the causative organism is very difficult with low yields.
- 2. Isolating the organism is generally difficult, unless there is coexisting abscess or suppuration. Needle aspiration and punch biopsy have poor yields. Nasal cultures have a role in confirming cases of community-associated MRSA, with a nasal colonization rate of *S aureus* of 28%, with 60% being community-associated MRSA.
- 3. Although blood cultures are recommended in patients with signs of bacteremia or in immunocompromised patients, they do not significantly change treatment for uncomplicated cellulitis in immunocompetent adults.

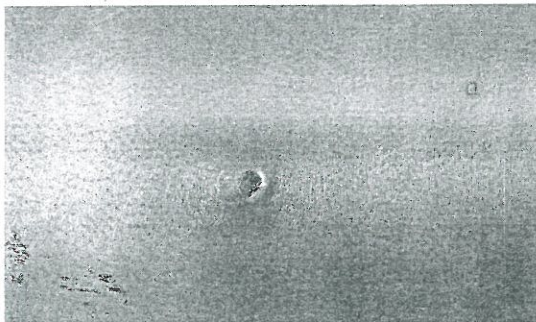
E. Management

1. The classic strategy includes analgesia, elevation, heat, and immobilization.
2. Any component of abscess formation requires incision and drainage.
3. Consider hospitalization for patients who are immunosuppressed or have facial involvement or multiple comorbidities.
4. Outpatient antibiotic therapy for simple cellulitis can include dicloxacillin, cephalexin, or a macrolide.
5. Inpatient antibiotic therapy includes parenteral first-generation cephalosporins (cefazolin) and penicillinase-resistant penicillins (nafcillin, oxacillin). In diabetic patients, use second- or third-generation cephalosporins (ceftriaxone) or imipenem if severe.
6. In cases in which community-associated MRSA is suspected, preferred antibiotics include TMP-SMX, clindamycin, tetracyclines, or vancomycin. Linezolid is another option for oral treatment but is somewhat cost prohibitive.
7. Erysipelas can be specifically treated with penicillin G; however IV nafcillin, a second- or third-generation cephalosporin, or amoxicillin-clavulanate should be used in diabetic patients and in patients with facial involvement. All patients with erysipelas should be considered for hospitalization and IV antibiotics.
8. Patients with necrotizing cellulitis require an early surgical consult, antibiotic treatment with penicillin G or clindamycin, and hospitalization.
9. For odontogenic source of facial cellulitis, treat with clindamycin or amoxicillin-clavulanate.
10. In cases of exposure to fresh, salt, or brackish water, treat with doxycycline, fluoroquinolones, or ceftazadime.
11. In cases of mammalian bites with cellulitis, treat with amoxicillin-clavulanate. If penicillin-allergic, treat with fluoroquinolone + clindamycin or TMP-SMX + metronidazole.

IX. CUTANEOUS ABSCESS

A. Epidemiology

1. A cutaneous abscess is a localized collection of pus with associated pain, fluctuant mass, and erythema.
2. Most abscesses (95%) contain bacteria, and the cause of the abscess varies depending on the location of the lesion.
3. While abscesses are usually localized, the presence of fever or other systemic symptoms suggests possible bacteremia.



Courtesy of David Effron, MD, FACEP

B. Pathophysiology

1. The development of cutaneous abscess depends on location. On extremities, the cause is usually minor trauma that damages the integrity of the epithelium. In intertriginous regions, abscesses are associated with obstructed apocrine sweat glands.
2. An abscess typically forms as a result of a lack of protective host factors or overwhelming bacterial contamination. Most start as a localized cellulitis with subsequent loculation of leukocytes and cellular debris with progressive liquefaction of the contents.
3. Etiology: the bacteria involved can depend on the location and predisposing factors.
 - a. Scalp, trunk, extremities: *Staphylococcus* spp, *S aureus* most common. A furuncle is a localized abscess associated with hair follicles. A carbuncle results if several furuncles coalesce and interconnect by sinus tracts; it may develop in areas of thick skin such as the back of the neck.
 - b. Intertriginous regions: *Escherichia coli*, *Proteus mirabilis*, and *Klebsiella* spp
 - c. Perirectal: mixed aerobic and anaerobic, *Bacteroides* most common
 - d. Foreign bodies: *S aureus*
 - e. Cat bites: *Pasteurella multocida*, *S aureus*, *S viridans*
 - f. Human bites: *Eikenella corrodens*, *Streptococcus*, *Staphylococcus* spp, *Fusobacterium*, *Peptostreptococcus*, *Corynebacterium* spp
 - g. IV drug abuse: mixed, predominant anaerobic, *Peptostreptococcus* most common, increased anaerobes in cocaine injections
4. Community-associated MRSA has emerged as the prevalent pathogen of skin and soft-tissue infections in urban areas. Furunculosis appears to be the strongest predictor of community-associated MRSA.

C. Management

1. A review of the literature found no significant difference in outcome of abscesses that were incised and drained and then treated with or without antibiotics, including those in which MRSA was considered to be the prevalent pathogen.
2. Current guidelines state that a cutaneous abscess can be effectively treated with incision and drainage alone; however, antibiotics are recommended in the setting of recurrent or persistent abscess, large abscesses (>5 cm), abscesses associated with surrounding cellulitis, abscesses associated with systemic symptoms, and abscesses in immunocompromised patients.
3. Proper identification of the abscess can be facilitated by examination, needle aspiration, and bedside ultrasonography. Ultrasonography can be used to localize fluid and measure dimensions of the abscess.
4. Preprocedural antibiotics are recommended in those at risk of endocarditis.
5. Local anesthetics can be less effective because of the acidic nature of infected tissue.
6. Proper incision and drainage technique includes sterile technique and universal precautions, an adequate size incision to create drainage, blunt dissection and irrigation of the cavity as needed, and packing of the wound. Any packing of the wound should be removed or changed in 2–3 days.
7. Large or deep abscesses should be treated in an operating room setting, and consultation should be considered in areas of cosmetic concern such as the face and breast, as well as other areas where complications can occur such as the palm, soles, or nasolabial fold.
8. For a discussion of antibiotic treatment, see cellulitis, page 1066.

X. STAPHYLOCOCCAL SCALDED SKIN SYNDROMES

A. Etiology

1. Exotoxin-producing *S aureus* of phage group 2
2. Actually a more severe form of bullous impetigo

B. Clinical presentation

1. Usually seen in children <2 years old (most commonly infants)
2. Often follows an upper respiratory infection or purulent conjunctivitis
3. Generally starts on the face (perioral area is classic), neck, axillae, and groin as erythematous patches that are tender to the touch. The erythema then spreads, becomes exfoliative, and is followed by development of flaccid bullae and skin desquamation. Crusting around the mouth and eyes is also frequently present. In newborns, the entire skin surface may be involved (Ritter disease).



Courtesy of David Efron, MD, FACEP

4. Mucous membranes are not involved, which helps differentiate from toxic epidermal necrolysis.
5. Tense pressure applied to the bullae may result in extension of the bullae because of an unstable epidermis; this is a positive Nikolsky sign, which is also seen in patients with toxic epidermal necrolysis and pemphigus vulgaris.
6. The cleavage plane is intraepidermal; therefore, only the superficial layers of the epidermis are shed, which also differentiates it from toxic epidermal necrolysis.
7. Lesions usually resolve in 2 weeks without scarring. Mortality rates in children are <5%; however mortality rates can be as high as 50% in adults and 100% in patients with severe underlying illness.

C. Management

1. Antibiotics to treat *Staphylococcus aureus*: β -lactamase/penicillinase-resistant penicillin such as oral dicloxacillin or IV oxacillin/nafcillin is the treatment of choice. Vancomycin or clindamycin may be considered if MRSA is a concern.
2. Steroids are contraindicated (they may exacerbate the illness).
3. Hospitalization for hydration and skin care is indicated for most patients (especially infants). Treatment is similar to that for thermal burn patients for wound care and rehydration with IV fluids.

XI. PEMPHIGUS VULGARIS

A. Definition, etiology, and epidemiology

1. An autoimmune bullous disease of skin and mucous membranes
2. A pathologic IgG attacks intercellular binding substance of epidermal keratinocytes (desmoglein) → acantholysis → extensive intraepithelial bullae formation
3. Most commonly affects patients 40–60 years old

B. Clinical presentation

1. Classic clinical scenario: A 55-year-old woman with a past medical history of lupus presents with a complaint of multiple fluid-filled bullae and painful, crusted ulcers. She has a recent history of several months of oral lesions of unknown etiology. Physical examination is significant for a positive Nikolsky sign.
2. Mucous membrane involvement (most commonly of the mouth) typically precedes development of cutaneous lesions by several months.
3. The cutaneous lesions are flaccid vesicles and bullae that rupture easily, leaving behind superficial erosions and crusted ulcerations. They are painful and can be seen anywhere.
4. Nikolsky sign is positive.
5. Mortality rate is currently 5%; however, before the use of corticosteroid therapy, this disease was almost uniformly fatal.

C. Diagnostic evaluation

1. Skin biopsy with Tzanck smear: a positive test (the presence of acantholytic epidermal cells with large nuclei in condensed cytoplasm) is suggestive but not specific.
2. Serum immunofluorescence and/or Dsg₃-ELISA: correlates loosely with the diagnosis.

D. Management

1. Dermatology consult
2. High-dose oral steroids (prednisone 2–3 mg/kg/day)
3. Concomitant immunosuppressive therapy (eg, azathioprine, methotrexate)
4. Antibiotics to treat secondary bacterial infection
5. Local therapy (eg, topical steroids)
6. Hospitalization for patients with extensive bullae and erosions
7. Other treatments include dapsone, IV immunoglobulin, and plasmaphoresis.

XII. HERPES INFECTIONS

A. Herpes simplex

See also herpetic whitlow, page 507; genital herpes, pages 569–571; and herpetic keratitis, pages 150–151.

1. Epidemiology

- a. Two variants: type 1 (HSV-1) and type 2 (HSV-2)
- b. Most HSV-1 infections involve nongenital areas: the lips (herpes labialis), the eyes (herpes keratitis), and the fingers (herpetic whitlow). However, HSV-1 is responsible for 10%–30% of infections involving the urogenital area.



Courtesy of David Efron, MD, FACEP

- c. Most HSV-2 infections involve the urogenital area: the vulva, vagina, cervix, and perineum in females and the penile shaft, glans penis, and anal area in males.



Courtesy of David Efron, MD, FACEP

- d. Spread of infection occurs through close contact with a person who is shedding the virus and involves direct contact of the vesicular fluid with mucous membranes or abraded skin.
- e. Incubation period for primary HSV infection is 2–20 days (average 6 days).
2. Clinical presentation
- The skin lesions begin as groups of vesicles (uniform in size) on an erythematous base that later rupture, ulcerate, and become crusted. The lesions develop at the site of inoculation, have a nondermatomal distribution, and are exquisitely tender.
 - Symptomatic primary HSV-2 infections are usually more severe than recurrent infections and are often accompanied by systemic signs and symptoms such as fever, malaise, myalgias, and regional adenopathy. Women with genital herpes often have urethral involvement and may present with severe dysuria and urinary retention.
 - Most primary HSV-1 infections are asymptomatic or mild (although they can be severe), but lesions may occur anywhere on the body.
 - In recurrent herpes (HSV-1 or HSV-2), a prodrome of local pain, burning, itching, and hyperesthesia usually precedes development of visible cutaneous lesions.
3. Diagnostic evaluation: Tzanck smear (multinucleated giant cells), viral culture (highest yield when lesions are vesicular), serology, immunofluorescence, and polymerase chain reaction (excellent for monitoring shedding).

4. Management

- a. Acyclovir decreases viral shedding, accelerates healing, and shortens the duration of symptoms but neither eliminates the herpes infection nor affects the frequency of recurrence.

- (1) Oral acyclovir is recommended for treatment of primary infections, particularly urogenital infections.

- (a) Dosage: 200 mg orally 5 times per day or 400 mg orally 3 times per day for 7–10 days or until clinical resolution

- (b) Famciclovir and valacyclovir appear to be efficacious and allow for less frequent dosing but are more expensive.

- (2) IV acyclovir is reserved for patients with severe disease or complications requiring hospitalization; dosage is 5–10 mg/kg IV every 8 hours for 5–7 days or until clinical resolution.

- (3) Although recurrent oral herpes generally requires no treatment, genital herpes may require episodic treatment. If used, oral acyclovir should be started at the onset of the initial prodromal symptoms or within 1–2 days after lesions appear.

- (4) Continuous daily prophylaxis with oral acyclovir is occasionally recommended for patients with frequent recurrences (>6 per year); it decreases the recurrence rate by 80%. It is also sometimes recommended for patients with uninfected sexual partners or who are immunosuppressed.

- b. Analgesia is often needed, particularly for primary infections. Topical viscous lidocaine is very effective, but oral agents (acetaminophen and hydrocodone, acetaminophen, and propoxyphene, etc) may also be required.

- c. Prompt ophthalmologic consult should be obtained for patients with ocular lesions.

- d. Counsel patients regarding the potential for recurrent outbreaks, asymptomatic viral shedding, and transmission to others; advise avoiding skin-to-skin contact when lesions or prodromal symptoms are present.

5. Complications

- a. Disseminated HSV can occur in neonates and adults with AIDS, malignancy, immunosuppression, or dermatitis, and may result in multisystem involvement.

- b. Eczema herpeticum refers to an acute infection of herpes simplex involving the skin lesions of atopic dermatitis. Lesions are typically vesicular but may umbilicate and ulcerate. Dissemination may occur. Most severe cases are in the very young or immunosuppressed and require hospitalization for IV antiviral therapy and bacterial superinfection.

B. Herpes zoster (shingles)

1. Epidemiology

- a. Herpes zoster is caused by reactivation of latent varicella-zoster virus (present since the initial infection with chickenpox).

- b. The elderly and immunocompromised are most commonly affected.

2. Clinical presentation

- a. Classic clinical scenario: A 77-year-old woman has a history of 3 days of constant right-sided chest pain in a band-like distribution. She reports recent increased stress from loss of a pet. Her past medical history is significant for having had varicella as a child. On examination, a vesicular rash in a dermatomal distribution is seen.

- b. Initially, pain or paresthesia develops at the dermatome (site of sensory nerve and ganglion) of future eruption. These symptoms usually precede the skin eruption by 3–5 days (range 1–21 days).
- c. This is followed by the development of herpetiform clusters of vesicles (which vary in size) on an erythematous, edematous base, with most of the vesicles concentrated on the proximal end of the dermatome.
- d. The lesions are almost always unilateral in distribution and limited to one or two contiguous dermatomes. The most commonly involved dermatomes are the thoracic (>50%), lumbosacral (10%–20%), and trigeminal (10%–20%).



Courtesy of David Effron, MD, FACEP

- e. **Hutchinson sign**
 - (1) Lesions at the tip of the nose signal possible eye involvement (the virus travels along the nasociliary branch of the trigeminal nerve, which is the ophthalmic division).
 - (2) Antiviral therapy is necessary to prevent blindness.
- f. **Ramsay Hunt syndrome:** facial palsy (similar to Bell palsy) associated with vesicles in the ear canal and on the pinna, tympanic membrane, and/or the pharynx (often associated with involvement of cranial nerve VIII)
- 3. **Diagnostic evaluation:** Tzanck smear (multinucleated giant cells), viral culture, or direct fluorescent antibody, which is more sensitive than culture and has a more rapid turnaround time
- 4. **Complications**
 - a. **Post-herpetic neuralgia:** more common in the elderly; risk is >40% in patients >60 years old.
 - b. **Dissemination** (more common in patients with Hodgkin disease)
 - c. **Bacterial superinfection**
 - d. **Reactivation or recurrence** (immunocompromised host)
 - e. **Aseptic meningitis and encephalitis**
 - f. **Delayed contralateral hemiparesis**
 - g. **Cranial and peripheral nerve palsies**
 - h. **Acute retinal necrosis** (may cause blindness in HIV patients)

5. Management

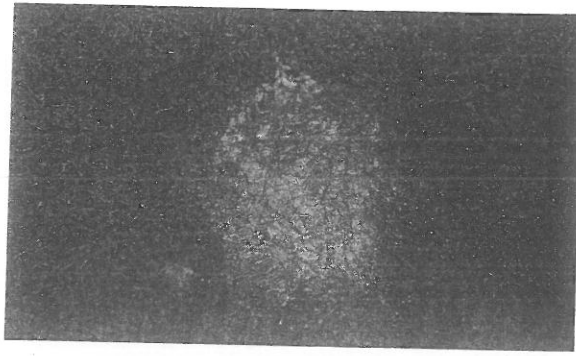
- a. Drying compresses (aluminum acetate, potassium permanganate)
 - b. Analgesics and referral to a pain-management specialist
 - c. For immunocompetent patients, oral antiviral therapy if it can be administered within 72 hours of the eruption.
 - (1) It speeds the healing of lesions, reduces the duration of acute pain, diminishes viral shedding, and decreases the duration (and possibly the frequency) of post-herpetic neuralgia.
 - (2) Valacyclovir and famciclovir are preferred to acyclovir because of their superior pharmacokinetic profiles and simpler dosing regimens.
 - d. For immunocompromised patients (including patients receiving steroids or chemotherapy), those with disseminated zoster, and those with eye involvement, use IV acyclovir.
 - (1) It decreases the rate of dissemination as well as morbidity in these patients.
 - (2) Dosage is 10 mg/kg every 8 hours for 5–7 days.
 - (3) These patients should be hospitalized.
 - e. Systemic corticosteroids: Although some authors believe these agents decrease the rate and severity of post-herpetic neuralgia in older patients when administered early, this benefit has not been confirmed in controlled studies; however, when used in combination with acyclovir, systemic corticosteroids improve quality of life during the acute infection.
 - (1) Combination therapy using valacyclovir or famciclovir with corticosteroids is assumed to be equally effective, but it has not been studied in clinical trials.
 - (2) Do not use corticosteroids in patients with diabetes or gastritis, because these drugs aggravate both conditions.
 - f. Immediate ophthalmologic consult is indicated if the ophthalmic branch of the trigeminal nerve is involved. (See also herpes zoster ophthalmicus and its management, page 151.)
6. Herpes zoster is a communicable disease; nonimmune contacts can develop chickenpox (varicella).
- a. Patients with herpes zoster should be advised to avoid contact with people who have not had chickenpox (the nonimmune), pregnant women, and immunocompromised patients.
 - b. Susceptible immunocompromised patients who are exposed should be given varicella-zoster immune globulin within 72 hours of exposure.

XIII. FUNGAL DISORDERS

A. Clinical presentation

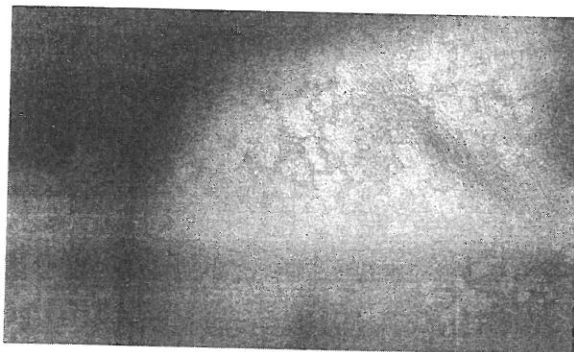
1. *Tinea capitis*

- a. A dermatophyte infection of the scalp characterized by areas of alopecia, broken hairs ("black dots"), and peripheral scaling



Courtesy of David Effron, MD, FACEP

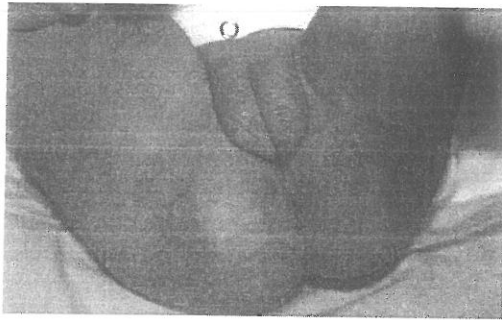
- b. Seen most commonly in children, with increased frequency among blacks
 - c. Uncommonly, an intense inflammatory response results in a boggy, tender, indurated plaque with pustules and alopecia referred to as a kerion. This lesion may result in permanent scarring and hair loss.
- 2. *Tinea barbae*
 - a. A dermatophyte infection of the beard area characterized by areas of plaques and patches; may also resemble bacterial folliculitis
 - b. Most common in adult men
- 3. *Tinea pedis*
 - a. A dermatophyte infection of the feet seen most often in young adult men
 - b. Three main types of infection: interdigital (most common), moccasin, and vesicular
- 4. *Tinea manuum*
 - a. A dermatophyte infection of the palmar surface of the hands
 - b. Characterized by dry scales and minimal inflammation
- 5. *Tinea cruris*
 - a. A dermatophyte infection of the groin common in adult men
 - b. Characterized by erythema with scaling borders of the inner thighs and buttocks, avoiding the scrotum and penis
- 6. *Tinea versicolor*
 - a. A superficial yeast infection caused by *Malassezia globosa*
 - b. Characterized by superficial scaling patches of various colors



Courtesy of David Effron, MD, FACEP

7. *Candida intertrigo*

- a. Infections that usually favor moist occluded areas such as skin folds and diaper areas
- b. Characterized by erythema and maceration as well as surrounding erythematous papules (satellites)



Courtesy of David Efron, MD, FACEP

- c. Predisposing factors include previous antibiotic or corticosteroid use, as well as immunocompromised states and obesity.

8. Sporotrichosis

- a. A mycotic infection caused by the fungus *Sporothrix* that is common in soil and plants
- b. Result of traumatic inoculation that results in a local inflammatory response involving lymphatics; however, dissemination may occur and result in extracutaneous illness.

B. Diagnostic evaluation

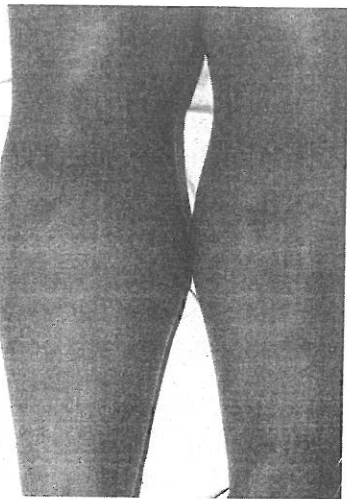
1. Diagnosis of dermatophyte infections and intertrigo are confirmed by identification of fungal elements on a potassium hydroxide preparation or a positive fungal culture; a positive examination usually reveals hyphae for diagnosis. Yeast such as *Candida* demonstrate pseudohyphae and spores.
2. Wood's light examination is of less help, because many dermatophytes do not fluoresce.

C. Management

1. Topical imidazole antifungal agents such as clotrimazole are indicated for most cutaneous fungal infections. Treatment should continue for an additional week after lesions have cleared.
2. *Tinea capitis*
 - a. Oral griseofulvin for 8 weeks
 - b. A topical shampoo containing ketoconazole is used to reduce contagiousness.
 - c. Kerion: oral prednisone is indicated as well as oral cephalexin for bacterial superinfection.
3. Sporotrichosis
 - a. Oral itraconazole
 - b. IV amphotericin B is reserved for disseminated cases.
4. *Tinea versicolor*
 - a. 2.5% selenium shampoo
 - b. Recurrence rates are as high as 50%.
5. All lesions should be kept clean and dry. Astringents such as 5% aluminum acetate aid in drying.

XIV. ERYTHEMA NODOSUM

- A. An inflammatory/immunologic reaction of the panniculus that most commonly affects women 15–30 years old
- B. Clinical presentation
 - 1. Characterized by appearance of painful, deep-seated nodules on the lower extremities
 - 2. These nodules are 3–20 cm in diameter, erythematous to violet in color, bilateral (but not symmetrical) in distribution, and tender on palpation. They are typically located on the anterior aspect of the tibia (particularly the shins) but can also be seen on the arms and (rarely) the face.



Courtesy of David Effron, MD, FACEP

- 3. Fever, malaise, and arthralgias (particularly of the ankles) often precede the cutaneous eruption.
- C. Predisposing factors (most cases are idiopathic or secondary to a recent streptococcal infection)
 - 1. Bacterial and fungal infections (eg, β -hemolytic streptococci, tuberculosis, tularemia, histoplasmosis, coccidiomycosis)
 - 2. Drugs (particularly birth control pills and sulfonamides)
 - 3. Leukemia, lymphoma, carcinoma
 - 4. Miscellaneous (eg, sarcoidosis, inflammatory bowel disease)
 - 5. Pregnancy
- D. Management
 - 1. Identify and treat underlying cause; this condition is self-limited (3–6 weeks) if the cause can be eliminated.
 - 2. Symptomatic measures
 - a. Bed rest and leg elevation
 - b. Compressive dressings
 - c. ASA or NSAIDs
 - 3. Corticosteroids may exacerbate underlying tuberculosis, lymphoma, or mycoses.

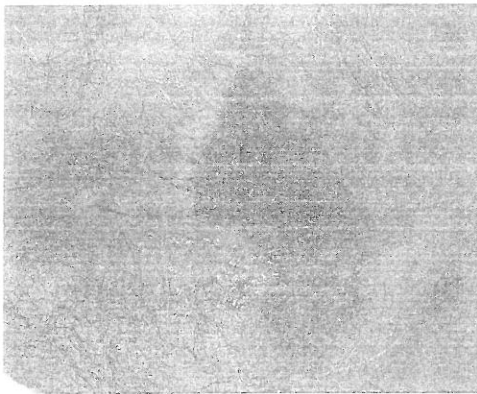
XV. DECUBITUS ULCER

A. Overview

1. The pressure of the individual's weight against hard surface causes tissue ischemia.
2. The normal instinct to shift positions is impaired by mobility issues, sedation, contraction/spasticity, or sensory loss.
3. Other contributors include malnutrition, anemia, comorbidities, and improper skin care.
4. The typical patient population includes the elderly, patients who are hospitalized or in an extended-care facility, and those with neurologic impairments.

B. Clinical presentation

1. Classic clinical presentation: The patient, a 67-year-old woman from an extended-care facility has severe multiple sclerosis and a fever. On examination, a large, malodorous, necrotic decubitus ulcer is noted over the sacrum. On probing, the tract extends to the sacrum. Results of laboratory studies include increased WBC count, erythrocyte sedimentation rate, and C-reactive protein. Radiographs reveal osteomyelitis.



Courtesy of David Effron, MD, FACEP

2. Occurs primarily on the hip/buttocks (70%) and lower extremities (25%)
3. Muscle necrosis occurs before skin breakdown; a small breakdown of skin may herald a large underlying cavity.
4. Stages of decubitus ulcers
 - a. Stage I: skin intact with erythema or ischemic changes
 - b. Stage II: partial-thickness loss of skin with blistering or superficial ulceration
 - c. Stage III: full-thickness loss of skin extending into subcutaneous tissue with crater-like appearance
 - d. Stage IV: full-thickness loss of skin with extension to muscle, bone, tendon, joint, or other deep structure; may be associated osteomyelitis

C. Management

1. Examine for odor, drainage, necrosis, other signs of infection; may need wound biopsy to differentiate contamination from infection.
2. Administer antibiotics for infection.
3. Mainstay of treatment is dressing, local wound care, and debridement.
4. Consult wound care team.

CUTANEOUS DISORDERS: QUICK REVIEW TIPS

Differential diagnosis of genital ulcers

- Chancre (primary syphilis) → *Treponema pallidum*
- Genital herpes → herpes simplex
- Chancroid → *Haemophilus ducreyi*
- Lymphogranuloma venereum → *Chlamydia trachomatis*
- Granuloma inguinale (donovanosis) → *Klebsiella granulomatis*

Wood's light

The Wood's light is useful in detecting certain organisms (listed below) because they fluoresce. It can give false-positive results when certain substances that fluoresce (eg, exudates, tetracycline in sweat, make-up, deodorant, and soap) are also detected. *Trichophyton tonsurans* (the most common cause of tinea capitis) does not fluoresce.

- Fungi that cause ringworm (*Microsporum audouinii*, *M canis*, *M distortum*)
- *Tinea versicolor* (may be imperceptible)
- Erythrasma
- *Pseudomonas aeruginosa*
- Tuberous sclerosis macules
- Porphyria cutanea tarda (urine or blister fluid)
- Hypopigmented skin

CUTANEOUS DISORDERS: PRACTICE CLINICAL SCENARIOS

Answers immediately follow the practice clinical scenarios.

Scenario A

Presentation: A patient presents with salmon-colored papules and plaques (sharply margined) covered with silvery white scales. Lesions are also found on the scalp, presacral area, knees, elbows, gluteal cleft, palms, soles, and extensor surfaces of the arms and legs. There is nail involvement with "pitting" and "oil spots."



Courtesy of David Efron, MD, FACEP

What is the diagnosis?

Scenario B

Presentation: A patient presents with erythematous papules and whitish "burrows" that are intensely pruritic and excoriated. Lesions are seen on the web spaces of the fingers, the flexion creases of the wrist and elbow, the penis, the buttocks, and the nipples. Pruritus intensifies at night.

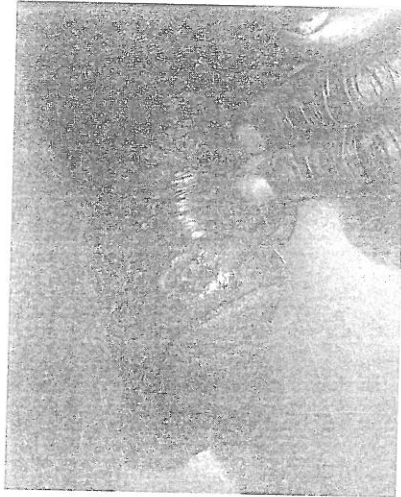


Courtesy of David Efron, MD, FACEP

What is the diagnosis?

Scenario C

Presentation: A patient has a painless, indurated ulcer with a smooth base and raised border on the genitals and another on the mucous membranes of the mouth (the site of inoculation). He also has a painless regional adenopathy.

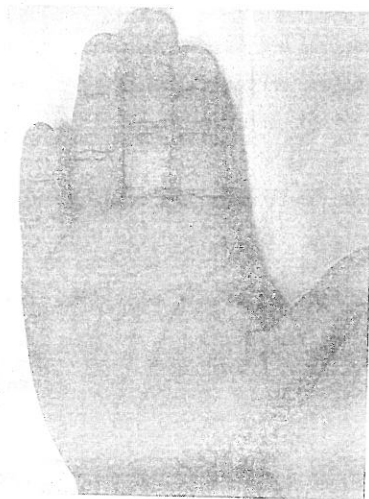


Courtesy of David Effron, MD, FACEP

What is the diagnosis?

Scenario D

Presentation: A patient presents with an erythematous or pink maculopapular or papulosquamous eruption with a symmetric distribution over the entire trunk and the extremities, including the palms and soles. Lesions are nonpruritic and accompanied by generalized lymphadenopathy and malaise.

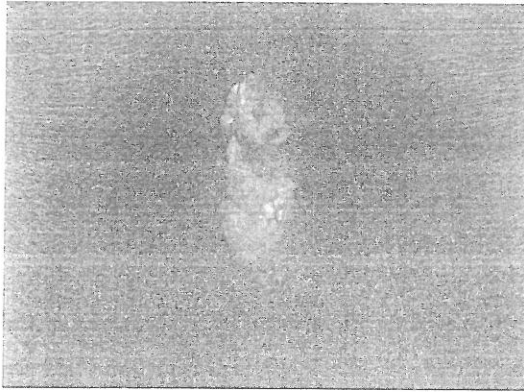


Courtesy of David Effron, MD, FACEP

What is the diagnosis?

Scenario E

Presentation: A patient presents with pedunculated, pale, cauliflower-like warts. The lesions are soft, moist, and painless condyloma lata.



Courtesy of David Efron, MD, FACEP

What is the diagnosis?

Scenario F

Presentation: A young, sexually active woman presents with tender, erythematous (or hemorrhagic) macules or papules that are evolving into pustules and vesicles with a "red halo" and a gray necrotic center. Lesions are few in number and have a predilection for the periarticular regions of the distal extremities. She also has a low-grade fever, chills, and migratory polyarthralgias.

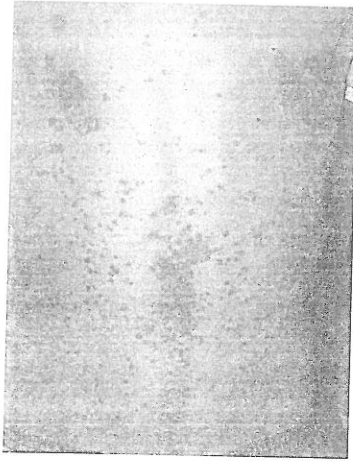


Courtesy of David Efron, MD, FACEP

What is the diagnosis?

Scenario G

Presentation: A teenager presents with oval-shaped, salmon-colored papules or plaques that have erupted on the trunk and proximal extremities. The lesions have a marginal collarette of scales, are mildly pruritic, and are distributed along the lines of skin cleavage, with the long axis of the lesions parallel to the ribs, forming a "Christmas tree" pattern on the trunk. On questioning, the patient indicates that he had a "herald patch" about a week ago.



Courtesy of David Effron, MD, FACEP

What is the diagnosis?

Scenario H

Presentation: A patient presents with a pink/red maculopapular rash that first appeared on the face, but then rapidly spread to the neck, trunk, and extremities, and is now fading by the third day.



Courtesy of David Effron, MD, FACEP

What is the diagnosis?

Scenario I

Presentation: An active well-appearing child has had a high fever lasting 3–4 days followed by eruption of an evanescent, blanching, rose-colored macular to maculopapular rash that starts on the trunk and spreads rapidly to the neck and proximal extremities. Mucous membrane involvement is absent. The fever breaks as the rash erupts.

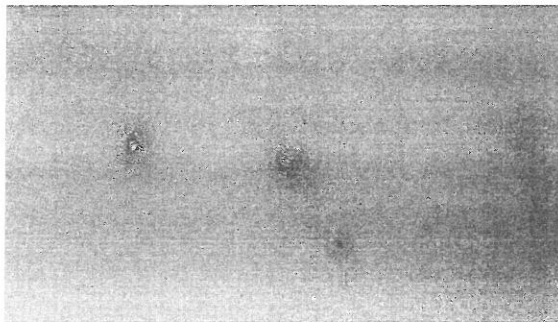


Courtesy of David Efron, MD, FACEP

What is the diagnosis?

Scenario J

Presentation: A patient presents with a vesicular skin eruption that started on the trunk and then spread to the face (including the mucous membranes of the mouth) and extremities. The palms and soles are spared. The vesicles (described as “dew drops on a rose petal”) rapidly evolve into pustules that umbilicate and crust. Successive crops of lesions continue to erupt over several days, so that lesions in all stages of development are present.

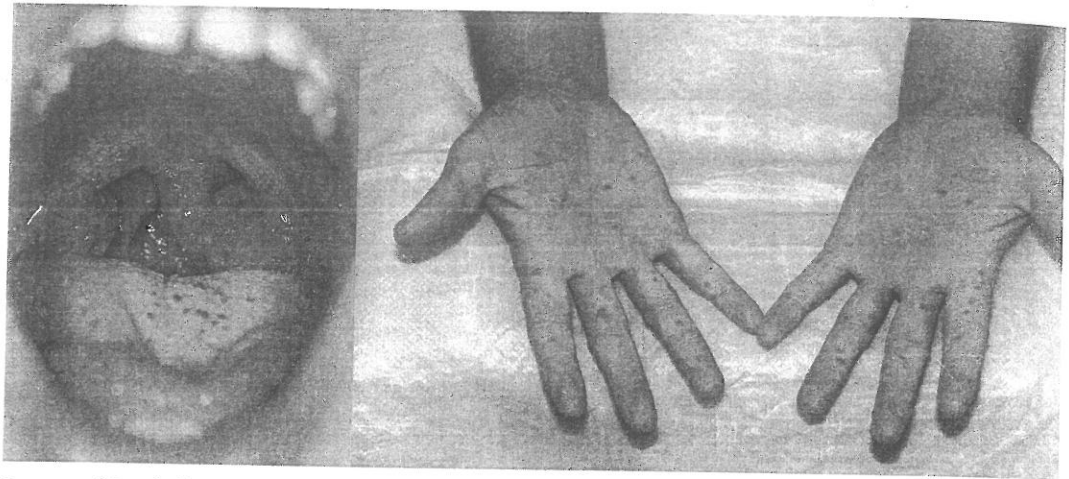


Courtesy of David Efron, MD, FACEP

What is the diagnosis?

Scenario K

Presentation: A patient presents with a tender vesicular rash that started on the oral mucosa and then spread a day later to involve the buttocks, hands, and feet (including the palms and soles). The vesicles are flat-topped and have an erythematous base.



Courtesy of David Effron, MD, FACEP

What is the diagnosis?

Scenario L

Presentation: A patient presents after having had a bright-red malar rash ("slapped cheek" appearance) for 2 days that has now developed into an erythematous, maculopapular rash that has spread to the trunk and limbs (the palms and soles are spared). The rash is fading with central clearing, creating a reticulated or lacy pattern.

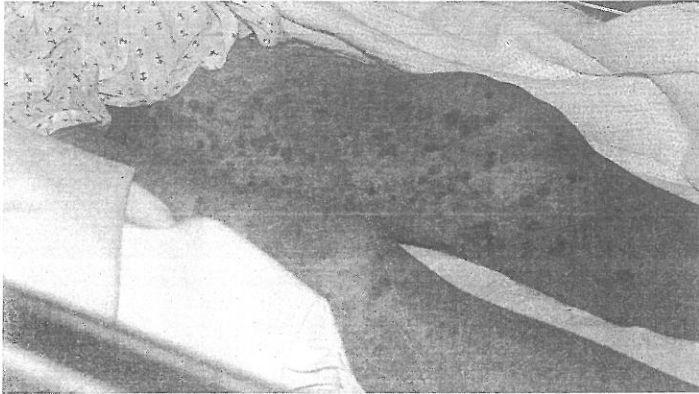


Courtesy of David Effron, MD, FACEP

What is the diagnosis?

Scenario M

Presentation: A patient presents with red to purple skin or mucous membrane lesions that do not blanch with pressure.



Courtesy of David Efron, MD, FACEP

What is the diagnosis?

Scenario N

Presentation: A patient reports mucous membrane hyperemia and a diffuse blanching, macular erythroderma (looks like a first-degree sunburn) that faded within 72 hours of its appearance. After another 1–2 weeks, there is now desquamation, particularly on the palms and soles. The patient has a fever ($\geq 38.9^{\circ}\text{C}$), hypotension, and involvement of three or more organ systems.



Courtesy of David Efron, MD, FACEP

What is the diagnosis?

Scenario O

Presentation: A patient presents with tender, shiny, erythematous plaques with raised and sharply demarcated borders on the face and lower extremities.

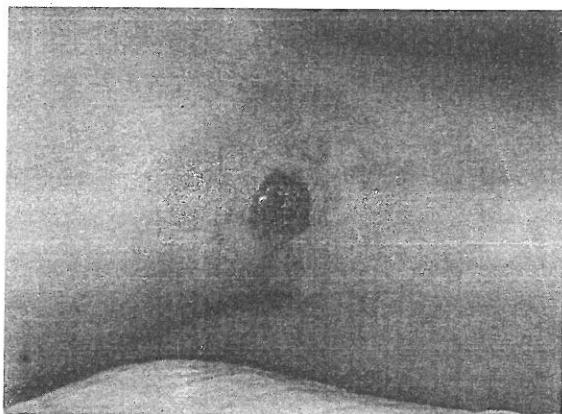


Courtesy of David Efron, MD, FACEP

What is the diagnosis?

Scenario P

Presentation: A patient presents with erythematous macules that have quickly evolved to bullae or pustules. The epidermis is sloughing, revealing an indurated, gunmetal gray, painless ulcer.



Courtesy of David Efron, MD, FACEP

What is the diagnosis?

ANSWERS TO CLINICAL PRACTICE SCENARIOS

Scenario A

Diagnosis: psoriasis

Management: Treatment options include topical steroids, UV light, coal tar-based shampoos, and immunosuppressants for severe cases; refer to dermatology.

Scenario B

Diagnosis: scabies (Be able to recognize a picture of the mite causing scabies.)

Management: Treat with scabicide (lindane or permethrin) and an antihistamine or topical steroid for pruritus. Treat household contacts and counsel on washing linens, etc.

Scenario C

Diagnosis: chancre of primary syphilis

Management: 2.4 million units penicillin G (1 dose) IM. Note that the average incubation period is 21 days but ranges from 10 to 90 days.

Scenario D

Diagnosis: secondary syphilis

Management: 2.4 million units penicillin G (1 dose) IM. Note that the rash usually erupts 2 weeks to 6 months (average 9 weeks) after the primary chancre appears.

Scenario E

Diagnosis: anorectal and genital warts, condyloma lata (a highly contagious manifestation of secondary syphilis); condyloma acuminata is caused by the human papilloma virus

Scenario F

Diagnosis: disseminated gonococcal disease

Management: Admit for treatment with IV ceftriaxone. Watch for complications, which include septic joint, meningitis, and endocarditis.

Scenario G

Diagnosis: pityriasis rosea

Management: Treatment is not necessary in most cases. Children can return to school with this rash.

Scenario H

Diagnosis: German or three-day measles (rubella)

Key facts:

- Caused by a genus of the rubivirus
- Incubation period of 14–21 days
- Period of infectivity extends from 7 days before until 7 days after onset of the rash.
- A prodrome of headache, malaise, sore throat, coryza, and low-grade fever is common in adults and adolescents 1–5 days before onset of the rash but is often absent or minimal in children.
- Forchheimer sign (pinpoint petechiae on the soft palate) may be present during the prodrome.
- Lymphadenopathy (suboccipital, postauricular, and posterior cervical) is characteristic.
- Complications include arthritis in adults (especially women), encephalitis, thrombocytopenia, and the congenital rubella syndrome when exposure occurs during the first trimester of pregnancy.

Scenario I

Diagnosis: roseola infantum (exanthem subitum)

Key facts:

- Caused by human herpesvirus type 6 and type 7
- Incubation period of 10–14 days
- Most commonly affects children 6 months to 3 years old
- Febrile seizures are a common complication.

Scenario J

Diagnosis: chickenpox (varicella)

Key facts:

- Caused by the varicella-zoster virus
- Incubation period of 14–21 days
- Period of infectivity is from several days before onset of the rash until all the lesions have crusted over.
- Prodrome of low-grade fever, malaise, and headache occurs in adults and adolescents 1–2 days before the rash appears.

- Diagnosis is usually made clinically but can be confirmed by Tzanck smear (multinucleated giant cells) or culture.
- Common complications include bacterial superinfection of the skin lesions, pneumonia, and encephalitis. Maternal infection during the first trimester can result in the congenital varicella syndrome. Perinatal maternal infection (5 days before to 2 days after delivery) can result in disseminated herpes in the neonate.

Scenario K

Diagnosis: hand-foot-and-mouth disease

Key facts:

- Caused by an enterovirus (usually coxsackie A16)
- Incubation period of 3–6 days
- Brief prodrome (if any) of low-grade fever, malaise, and sore mouth
- Infection during the first trimester of pregnancy may result in spontaneous abortion.

Scenario L

Diagnosis: fifth's disease (erythema infectiosum)

Key facts:

- Caused by human parvovirus B19
- Incubation period of 4–14 days
- May be accompanied by fever, headache, malaise, and myalgias; in adults (particularly women), arthralgias and arthritis are also common.
- May recur with various stimuli over several months
- Once rash appears, children are generally no longer contagious.
- Complications are rare unless the infection occurs during pregnancy (which may cause hydrops fetalis or fetal death), or they may occur in patients with chronic hemolytic anemias (which may cause aplastic crisis).

Scenario M

Diagnosis: purpura

Key facts:

- Purpuric lesions <3 mm are referred to as petechiae, while larger lesions are called ecchymoses. These lesions are due to extravasation of blood in the dermis.
- Nonpalpable purpura occurs in association with conditions that produce thrombocytopenia, nonfunctional platelets, or coagulation defects. Examples include idiopathic thrombocytopenic purpura, thrombotic thrombocytopenic purpura, aplastic anemia, and the hemophilias.
- Palpable purpura signifies dermal inflammation caused by a vasculitis (infectious or immune-mediated). Henoch-Schonlein purpura is a classic example.
- Petechiae/purpura in the presence of fever suggests bacterial infection (bacteremia, sepsis, or meningitis): *Neisseria meningitides*, *Streptococcus pneumoniae*, *Neisseria gonorrhoeae*, *Haemophilus influenzae*, *Staphylococcus aureus*.

Scenario N**Diagnosis:** toxic shock syndrome**Key facts:**

- Menstrual-related toxic shock syndrome occurs in young women and is associated with the use of high-absorbency tampons for prolonged periods of time. Risk factors for nonmenstrual-related toxic shock syndrome are osteomyelitis, abscesses, insulin pump site, surgical and nonsurgical wounds, nasal packs, septic abortion, postpartum infections, and the use of contraceptive sponges or diaphragms.
 - More than 50% of severely ill patients experience hair and nail loss 2–3 months after the initial insult.
 - Toxic shock syndrome is a toxin-mediated illness; most cases (>90% of menstrual-related and ~60% of nonmenstrual-related) are caused by strains of *Staphylococcus aureus* that produce toxic shock syndrome toxin 1.
 - Although prevalent in the 1980s, the number of cases of menstrual-related toxic shock syndrome has declined sharply and currently represents only 50% of all toxic shock syndrome cases.
 - Treatment includes early antibiotics (clindamycin preferred), removal of source, and aggressive resuscitation.
-

Scenario O**Diagnosis:** erysipelas**Key facts:**

- This variety of cellulitis (caused by group A streptococci) involves only the upper dermis. Traditional cellulitis has a much less distinct border and involves the deep dermis and subcutaneous fat.
 - Incubation period of several days
 - Infants, young children, and the elderly are most commonly affected.
 - Associated signs and symptoms (high fever, chills, and anorexia) develop rapidly and may precede the appearance of the cellulitis by 1–2 days.
 - Appropriate antibiotics include penicillins, cephalosporins, and macrolides.
-

Scenario P**Diagnosis:** ecthyma gangrenosum

Management: Admit for IV antibiotics; choices include broad-spectrum penicillins such as piperacillin, third-generation cephalosporins such as cefepime, aminoglycosides, and fluoroquinolones. Most common cause is *Pseudomonas aeruginosa*, with >50% mortality when there is an associated bacteremia.

EMERGENCY MEDICAL SERVICES

Background Information	1096
Definition	1096
Origin of the Current EMS System.....	1096
Models of EMS Service/System Design.....	1098
Planning for EMS System Demands	1098
Components of an Emergency Medical Services System	1099
Prevention	1099
Manpower	1099
Education and Training of EMS Providers	1099
Communications	1100
Hospital Emergency Department and Specialty Service Categorization Program	1101
Medical Control	1101
Transport	1101
Medical-Legal Issues.....	1102
Occupational Issues for EMS Providers	1103
Aeromedical Transport.....	1103
Disaster Planning and Operation	1104

EMERGENCY MEDICAL SYSTEMS: SELF-ASSESSMENT QUESTIONS

1. The type of transportation vehicle selected is determined by all of the following except:
 - (a) The level of medical training of the medical care provider
 - (b) The distance and time needed to reach the receiving hospital
 - (c) The patient's condition
 - (d) The weather conditions and terrain that needs to be traversed
2. The major role of the hospital during a disaster operation is:
 - (a) Triage, stabilization, and transportation
 - (b) Providing scene-response medical teams
 - (c) Assuring the safety of emergency personnel, victims, and bystanders
 - (d) Medical care of disaster victims and the ongoing routine medical needs of the community
3. The primary goal of triage during a disaster operation is to:
 - (a) Treat victims as quickly as possible
 - (b) Do everything medically possible for each disaster victim
 - (c) Treat as many patients as possible with the given resources
 - (d) Classify victims according to treatment priorities to do the most good for the greatest number of potential survivors
4. During a disaster operation, patients who are unlikely to survive are classified by the triage color:
 - (a) Red
 - (b) Yellow
 - (c) Green
 - (d) Black
5. In order to be in compliance with The Joint Commission, all hospitals must have a written disaster plan and conduct disaster drills:
 - (a) One time per year
 - (b) Two times per year
 - (c) Three times per year
 - (d) Disaster drills are recommended but are not required, because they disrupt normal hospital operations.
6. An on-scene physician wants to assist advanced life support providers at the scene of an emergency call. The physician is recommending treatment that may differ from local EMS operational protocols. The EMS providers should take the following action:

- (a) Permit the on-scene physician to carry out interventions outside the scope of the provider's practice
 - (b) Defer all decisions about patient care to the on-scene physician
 - (c) Refuse to relinquish care unless the physician provides verification of current licensure
 - (d) Request that the physician accompany the patient and providers to the hospital and document all physician-performed interventions
7. Which of the following is true regarding aeromedical transport?
- (a) There is research to support use of rotary wing transport for all critically ill patients.
 - (b) Ground transportation is a better option for travel up to 30 miles or 30 minutes.
 - (c) Cost should not be considered in making the decision to use a specific transport method.
 - (d) When used for medical transport, fixed-wing aircraft fly at lower than standard altitudes to avoid problems such as air embolism and hypoxemia.
8. Which of the following EMS providers can give oxygen, transport a patient, and assist with administration of a patient's own prescribed medications?
- (a) Emergency Medical Technician
 - (b) Advanced Emergency Medical Technician
 - (c) Emergency Medical Responder
 - (d) Emergency Medical Technician-Intermediate
9. An ambulance transporting a critically ill trauma patient arrives unexpectedly at your emergency department without prior consultation with medical control. The patient requires rapid administration of blood products and placement of a chest tube. What are the most appropriate next steps?
- (a) Refuse to accept responsibility for the patient, because the providers failed to consult and your emergency department does not have sufficient resources.
 - (b) Stabilize the patient to the best of your ability, and instruct the ambulance to proceed to the nearest trauma center.
 - (c) Perform the necessary lifesaving and stabilizing interventions, and ensure that the ambulance that will transfer the patient to the accepting trauma center has personnel trained to assess and monitor for emergency interventions.
 - (d) Have an emergency department nurse accompany the patient to the nearest trauma center.

ANSWERS

- | | | |
|------|------|------|
| 1. a | 4. d | 7. b |
| 2. d | 5. b | 8. a |
| 3. d | 6. d | 9. c |

Use the pre-chapter multiple choice question worksheet (page xx) to record and determine the percentage of correct answers for this chapter.

I. BACKGROUND INFORMATION

A. Definition

1. The spectrum of emergency medical care is known as the emergency medical services (EMS) system.
2. The EMS system began in the 1960s as an extension of emergency medical care into the community. Today, there are three distinct phases within the spectrum of the EMS system.
 - a. Prehospital care
 - b. Emergency department care
 - c. In-hospital care

B. Origin of the current EMS system

1. Highway Safety Act of 1966
 - a. Established the Department of Transportation and gave it legislative and financial authority to improve EMS, with each state required to develop regional EMS systems to handle their specific prehospital needs
 - b. Emphasis was placed on developing highway safety programs, EMS standards, improvement of ambulance service, and provider training.
 - c. Matching funds from state and local government targeted millions of dollars toward EMS research and development in the 1970s and 1980s.
2. Emergency Medical Services Act: initiated in 1973 and provided funds for the development of local, state, and regional EMS systems; included development of guidelines for emergency medical care
 - a. State EMS system
 - (1) The central EMS authority with ultimate responsibility for planning, implementing, and operating the state EMS system.
 - (2) May delegate authority to regional, county, and local authorities
 - b. Established 15 essential components of an EMS system
 - (1) Manpower
 - (2) Training
 - (3) Mutual aid
 - (4) Disaster plan
 - (5) Facilities
 - (6) Transportation
 - (7) Access to care
 - (8) Communications
 - (9) Critical care units
 - (10) Public safety agencies
 - (11) Consumer participation
 - (12) Patient transfer
 - (13) Public info/education
 - (14) Review and evaluation
 - (15) Coordinated patient record keeping

3. Establishment of communication and ambulance standards
 - a. The Department of Transportation and White House Office of Telecommunications advocated for national standard emergency access (911).
 - b. The Federal Communications Commissions established rules and regulations for EMS communications and dedicated specific radio frequencies for emergency systems.
 - c. The Department of Health, Education and Welfare delineated optimal ambulance design as well as essential equipment recommendations.
 - d. The General Services Administration issued federal specifications for ambulances, referred to as the ambulance KKK-A-1822F standards.
4. Establishment of industrial and EMS-related standards developed by the National Highway Transportation Services Administration's Emergency Services Bureau include the following:
 - a. EMS curriculum
 - b. Medical aircraft (rotary and fixed-wing)
 - c. EMS system organization
5. Trauma Care Systems Planning and Development Act of 1990 encouraged further EMS development and set a standard for directing patients to an appropriate designated facility for time-sensitive treatment.
6. EMS Agenda for the Future, first published in 1996, developed by a multidisciplinary group, outlined the current state of EMS and posted future goals with emphasis on improving system efficiency, utilizing technology, developing healthcare networks that include EMS, and engaging in public health activities.
 - a. An "Implementation Guide" set forth clear goals and objectives, along with interim steps to ensure their attainment.
 - b. The EMS Education Agenda for the Future (June 2000) revised the National Standard Curriculum.
 - c. The most recent Agenda calls for an updated scope of practice model. The scope of practice model is consistent with training paradigms found in other allied health professions. Nationally recognized provider levels include:
 - (1) Emergency Medical Responder (formerly "First Responder")
 - (2) Emergency Medical Technician (formerly Emergency Medical Technician-Basic)
 - (3) Advanced Emergency Medical Technician (formerly EMT-Intermediate)
 - (4) Paramedic (formerly "EMT-Paramedic")
 - d. The EMS functions at the federal level are overseen in the Department of Transportation and National Highway Traffic Safety Administration office of EMS.

Table 46: Comparison of Nationally Recognized Emergency Medical Services Providers

	Emergency Medical Responder	Emergency Medical Technician	Advanced Emergency Medical Technician	Paramedic
Focus	Initiates life-saving care	Provides emergency care and transportation	Provides basic and limited advanced life support care	Provides advanced level care to critically ill and injured; provides link from the scene to the emergency health care system
Skill set	Noninvasive airway management, hemorrhage control, use of automated defibrillator	Basic life support skills, oxygen administration, patient transport	Limited medications, airway management, IV fluids	Full complement of advanced cardiac life support medications, advanced airway management, advanced patient assessment

C. Models of EMS service/system design

1. Fire service-based EMS model: Under this design, the fire department provides all EMS services.
2. "Third service" model: In this model, the EMS delivery system is housed in a specially formed municipal department (separate from police and fire services) and provides basic life support (BLS) and advanced life support (ALS) services using municipal owned, operated and staffed ambulances.
3. "Public utility" model: In this model, a community contracts with a private ambulance company to provide BLS and ALS ambulance services for all emergency calls in their community.
4. Volunteer model: BLS, and in some systems, ALS ambulance service is provided by unpaid public service personnel.
5. Combination models: This service model combines different first-response and transport agencies.

D. Planning for EMS system demands

1. EMS providers should anticipate 100 service requests for EMS annually per 1,000 population in the area they are serving.
2. In service areas with a high volume of elderly or medically underserved, the call volume will be greater.
3. The EMS Agenda for the Future encourages collaboration with emergency health care services and public health agencies to participate in disease surveillance and injury prevention.
4. Currently, the Centers for Medicare and Medicaid Services (CMS) sets levels of reimbursement for patients transported by emergency medical services. Different rates apply for basic, advanced, and "specialty" care transportation.

II. COMPONENTS OF AN EMERGENCY MEDICAL SERVICES SYSTEM

A. Prevention (public education)

1. Safety and accident prevention
2. Vehicle safety and protective equipment
3. Poisoning prevention (including "childproof medications")
4. Education in schools
5. CPR, first aid, and automated defibrillator training

B. Manpower

1. 70%–80% of emergency medical technicians (EMTs) in the United States are volunteers.
2. The percentage of volunteers is highest in rural areas.

C. Education and training of EMS providers

1. In previous years, an emergency medical responder's scope of practice was defined by the National Standard Curriculum. Future EMS certification examinations and provider designations will be based on the National Scope of Practice EMS provider level. Although classifications are similar, the Scope of Practice model attempts to standardize training requirements.
2. Emergency Medical Responder (formerly "First Responder")
 - a. Basic life support skills
 - b. Hemorrhage control
 - c. Scene safety and vehicle extrication techniques
3. Emergency Medical Technician (formerly "Emergency Medical Technician–Basic")
 - a. Includes skill set and knowledge base of the emergency medical responder
 - b. Automated external defibrillation
 - c. Patient-assisted medication delivery (nitroglycerin, albuterol)
4. Advanced Emergency Medical Technician (formerly "Emergency Medical Technician–Intermediate")
 - a. Includes EMT skills
 - b. Supraglottic airway management
 - c. Limited medications (epinephrine for anaphylaxis, narcotic antagonist)
 - d. Patient-assisted medication administration
5. Paramedic (formerly "Emergency Medical Technician–Paramedic")
 - a. Includes Advanced EMT skills
 - b. Training in differential diagnosis, medical decision making
 - c. Invasive airway management
 - d. Advanced patient assessment skills
 - e. Licensure requirement includes graduation from nationally accredited certificate or associate's degree program
6. States may designate specialty levels of emergency medical services providers according to regional needs. Other commonly encountered "specialty" providers include:

- a. Critical care paramedic: EMS professional trained to the "paramedic" level and credentialed to provide interventions outside the typical scope of practice. Critical care providers may be trained to manage ventilators, administer blood, or perform advanced skills
- b. Advanced practice or community paramedic: In some regions, paramedics function as physician extenders. Advanced practice paramedics may collaborate with local health departments to conduct follow-up visits and monitor medications.

D. Communications

1. The purpose of a communications system is to provide a means of accessing and coordinating the EMS system.
2. Basic elements of a communications system
 - a. Discovery and response
 - (1) Recognition of the need for emergency notification
 - (2) Best resuscitation outcome is possible when BLS is started within 4 minutes of arrest and ALS is started within 8 minutes of arrest.
 - b. Access (the ability to activate the EMS system)
 - (1) A single telephone number (911) to access police, fire, and EMS is preferred over multiple seven-digit phone numbers, which may be difficult to locate/recall in a crisis.
 - (2) An enhanced/augmented 911 system (E-911) is a computerized system that automatically displays the address and phone number of the 911 call's origin to the emergency medical dispatcher.
 - (3) New system elements are present for automatic location of cell phone calls, which now account for a large percentage of 911 calls.
 - c. Receiving and dispatch
 - (1) Some EMS systems have separate 911 access/receiving and dispatch functions.
 - (2) The goal is to dispatch the most appropriate personnel and equipment to the location; initiating a maximal response (lights and sirens) for all requests is ineffective in terms of manpower (and financial resources) and can result in unnecessary harm to EMS providers and the public because of EMS vehicle accidents.
 - (3) The emergency medical dispatcher is responsible for:
 - (a) Taking and triaging the call
 - i. Relevant information about the emergency is ascertained first (including verification of location, nature of the call, and number of people involved).
 - ii. The dispatcher prioritizes the call as requiring a maximal or prompt response.
 - (b) Alerting and dispatching the appropriate unit
 - (c) Ensuring that prehospital personnel can find the address
 - (d) Providing prearrival instructions on how to assist the victim until the ambulance arrives
 - (e) Some systems have developed alternative call-management systems for 911. This allows low-priority calls to be managed over the phone or by using nonemergency resources. Alternative call-management systems may include the use of registered nurses or other professionals to provide medical-related advice via telephone.
 - d. Hospital notification and participation

- (1) EMT crews may communicate with medical control (on-line medical direction), which may or may not be the receiving hospital.
- (2) An advantage of hospital notification is that it allows time for emergency department personnel to mobilize the appropriate resources for patients' needs (particularly when an incident involves multiple casualties).

E. Hospital emergency department and specialty service categorization program developed by CMS)

1. Type A: fully equipped and staffed emergency department with equipment and personnel available 24/7, either with a license or accepted by the public as able to treat patients emergently
2. Type B: licensed by the state as an emergency department, held out to the public by posted signs, seeing at least one-third of visits on an urgent basis without appointment (Type B emergency departments are paid at a lower rate than Type A.)

F. Medical control

1. Physician input and surveillance assures medical competence of an EMS system. Medical control can be the responsibility of one physician, a group of physicians, or a hospital (referred to as the "resource hospital").

2. Components

a. Off-line (indirect) medical control

- (1) **Development of procedural/treatment protocols**
- (2) **Provision of standing orders when contact with medical control is not feasible in a timely fashion**
- (3) **Training and testing**
- (4) **Ongoing education and surveillance**
- (5) **Quality reviews and improvement**

b. On-line (direct) medical control

- (1) **Provision of direct medical orders to EMTs in the field**
- (2) **Direct in-field observation: the most effective method for assessing the quality of care provided by EMS personnel; provides the most complete data gathering as well as prompt feedback**

G. Transport

1. Vehicle standards (physical and medical) and standards for essential equipment are established jointly by government agencies and medical specialty groups.
2. The cost of an EMS ambulance varies according to its function and design. For example, ambulances providing rescue services (extrication) in addition to patient care may cost in excess of \$200,000.
 - a. Type I ambulances have a conventional pick-up chassis with a modular compartment to carry equipment, personnel, and the patient. There is no passageway between the driver and patient compartment.
 - b. Type II ambulances are van-type vehicles; the body and cab form a single unit, and most models have a raised roof for extra stand-up space.
 - c. Type III ambulances have a large chassis with a forward cab that is connected by a walk-through passage to the patient-care compartment.

3. The type of transport used is determined by the distance and time needed to reach the hospital, the patient's condition, the weather, and terrain. The following guidelines are helpful in determining the optimal use of each mode of transportation.
 - a. Ground transportation: best choice for distance ranges up to 30 miles or a transport time <30 minutes
 - b. Rotary-wing transportation: most effective range is 50–150 miles; best choice when terrain does not allow for use of ground vehicles, transit time would be delayed because of traffic congestion, or the distance to the hospital is too great; poor weather conditions limit its use. Rotary-wing (helicopter) transport is commonly used for victims of penetrating trauma who may be located far away from a designated trauma center.
 - c. Fixed-wing transportation: most effective for distances >100 miles; use may be limited by weather conditions, runways of inappropriate length/condition, refueling requirements, and altitude problems (hypoxemia, air embolism, expansion of air within catheter or endotracheal balloons).

H. Medical-legal issues

1. Consent to care is same as described for the emergency department
2. Refusal of care
 - a. A potentially significant legal problem (>50% of litigation against EMS providers is related to the issue of failure or refusal to treat or transport a patient who requests treatment or transportation).
 - b. A competent, conscious adult may make an informed decision to refuse treatment or transportation.
 - (1) The decision whether the patient is competent may be made in conjunction with the on-line medical control physician.
 - (2) The patient is informed of the risks involved in refusing care.
 - (3) The refusal should be well documented, and a release of liability waiver completed.
 - c. An incompetent patient (as determined by the EMS on-scene providers with or without EMS physician involvement) should not be allowed to refuse care. (Appropriate treatment and transportation should be provided, even if the patient physically resists it. Police may need to be called to the scene to help manage such patients.)
3. On-scene physician
 - a. An on-scene physician who is unknown to EMS providers must be able to provide proof of identity and medical licensure before being allowed to provide patient care guidance.
 - b. An on-scene physician may assist with treatment that is in line with EMS protocols without assuming responsibility and being required to accompany the patient to the hospital.
 - c. No physician, including the on-scene physician, can direct EMS providers to administer emergency care and treatment that is beyond the EMS providers' training and state regulations.
 - d. The on-line medical control physician may relinquish responsibility for guiding medical care of a patient to the on-scene physician; if the on-scene physician wishes to assume medical control, he or she must accept full medicolegal responsibility for all patient care and should accompany the patient to the hospital.

I. Occupational issues for EMS providers

1. Infectious disease risk
 - a. EMS personnel should observe universal precautions with every patient encounter because of risk of exposure to blood-borne pathogens.
 - b. EMS personnel should also observe appropriate precautions for disease-specific risks (eg, meningitis, HIV, tuberculosis, etc)
2. Occupational risks
 - a. Slips, falls
 - b. Lifting injuries
 - c. Assault
3. Legal risk
 - a. Malpractice risk: 1 claim/24,000 EMS calls
 - b. Operational risk: areas of liability include vehicle accidents, patient injury, medication errors, etc.

J. Aeromedical transport

1. Interfacility transports account for a larger percentage of all hospital-based transport flights versus scene responses.
2. Aeromedical transport of severely injured victims from a trauma scene has been shown to reduce mortality. Its effectiveness in reducing the morbidity and mortality of nontrauma patients, however, has not yet been clearly established.
3. The Federal Aviation Administration (FAA) regulates the aviation component (aircraft, flight crews, maintenance) of air medical services; specific FAA regulations apply only to EMS aircraft (not to in-flight patient care). Because of multiple aircraft crashes and fatalities, the FAA is increasing industry regulation.
4. The Commission on Accreditation of Air Medical Services, established in 1990, was developed to create a national voluntary accreditation process for air medical services. In 1997, it changed its name to the Commission on Accreditation of Medical Transport Systems (CAMTS). Accreditation standards were extended to address critical-care ground transport teams. While there are no regulatory implications, several managed care companies, state EMS agencies, and federal organizations are using CAMTS standards for benchmarking quality services as a prerequisite for contractual relationships and a requirement for state licensure.
5. Helicopter (rotary-wing) transport programs
 - a. Many rotary-wing programs permit flight only under visual flight rule conditions (clear weather). Many programs mandate that all aeromedical aircraft be equipped for instrument flight rule conditions to allow safe landing should poor weather develop en route.
 - b. Recent increases in the rate of fatal helicopter accidents have motivated discussions about rotary-wing safety. A decision to use helicopter transport should be predicated on:
 - (1) A thorough understanding of the patient's condition
 - (2) Associated hazards, including weather and risk to personnel
 - c. Helicopter emergency medical services have significant costs associated with operation and transport. Costs to the patient for an aeromedical evaluation can exceed \$6,000.

6. Airplane (fixed-wing) transport
 - a. Fixed-wing aircraft have a better safety record than helicopters; they are able to fly under a wider range of weather conditions and are generally less expensive to operate.
 - b. The cost for transport by fixed-wing aircraft ranges in the thousands, depending on the aircraft used and the distance flown.
 - c. Airplanes also provide an increased mile range; greater speed; and increased patient, crew, and equipment capacity. However, they are limited to areas with airports.

III. DISASTER PLANNING AND OPERATION

A. Definition of a medical disaster

1. An incident that results in multiple casualties, overwhelming numbers, or occurring at a rate that cannot be handled by the community resources
 - a. Natural disasters (hurricanes, floods, earthquakes, etc)
 - b. Human disasters (crashes [air, train, water], explosions, fires, environmental contamination, riots, terrorism, etc)
2. The key issue is not the absolute number of victims but the relationship between the needs of the victims and the ability of the health care system to meet those needs using normal operating procedures.

B. Disaster classification: disasters are most often classified according to the ability of the community, region, or state to meet the needs of the disaster response.

1. Level I disaster: local medical resources are adequate and quickly mobilized; declared by local officials.
2. Level II disaster: requires medical resources from adjacent communities and regions (mutual aid); mobilization of such additional resources may take several hours to a day.
3. Level III disaster: requires state or federal resources; requested by a local official, but actually declared by the governor or president; mobilization of these resources may take 2–3 days.

C. Creating a disaster plan

1. Characteristics of a disaster plan
 - a. Must comply with the National Response Framework (NRF); the NRF document, distributed by the Federal Emergency Management Agency, outlines guiding principles to follow in the event of a disaster. The goal of the NRF is for agencies involved in emergency preparedness to follow uniform disaster management strategies.
 - b. Must be simple but flexible enough to accommodate the type and size of the disaster
 - c. Must be as closely aligned with normal daily operating procedures as possible
 - d. Must be coordinated with adjacent geopolitical areas (mutual aid agreements)
 - e. Must include input and roles of local emergency management agencies, law enforcement, fire, public health departments, ambulance services, EMS council, hospitals, and the local medical community
 - f. Must be organized with a structure including the Incident Command System (ICS) and the National Incident Management System

- (1) Incident command functions are unified under a designated "Incident Commander."
- (2) **The ICS is a standardized, yet flexible template for local disaster operations:**
- (3) The ICS is further subdivided into four major "sections." Each section has specific responsibilities.

Table 47: Key Components of the Incident Command System (ICS)

Operations	Planning	Logistics	Finance
Search and rescue activities	Collect and manage incident-related data	Provide necessary support for incident operations	Manage financial aspects of operation
Tactical operations	Formulate incident action plans	Facilities	Track responder time and pay
Treat and transport injured	Conduct required meetings	Transport of supplies	Ensure ongoing financial support and distribution of funding to branches/sections
Triage of injured	Ensure adequate and transparent flow of information between sections	Equipment maintenance and fueling Food and medical services for responders	

- g. Courses on incident management free of charge to health care providers are available on the Federal Emergency Management Agency website (<http://www.fema.gov/incident-command-system#item7>).
- h. Must include a joint public information operation
2. Essential phases of a disaster plan
 - a. Activation
 - (1) Notification and initial response: The initial EMS personnel arriving on the scene report on the nature of the incident, extent of damage, the estimated numbers and types of injuries, hazards for the victims and rescuers, and the best access to the scene or routes that are known to be blocked. Activation is rapid (within minutes).
 - (2) Organization of an incident command post by senior fire/emergency response officials. A more in-depth assessment of the scene and associated hazards takes place after command post set-up.
 - b. Implementation
 - (1) Search and rescue (fire/rescue operation of identifying victims)
 - (2) Triage, stabilization, and transport (medical providers)
 - (3) Definitive management of scene hazards (firefighters) and victims (medical providers)
 - (4) Hospital activities during field operations
 - (a) Alert all area hospitals
 - (b) Define levels of care needed
 - (c) Monitor the status of the scene
 - c. Recovery
 - (1) Scene withdrawal (includes a systematic recheck for any missed victims)
 - (2) Return to normal operations (includes restocking)
 - (3) Debriefing
 - (a) Evaluation of the disaster response
 - (b) Identification of psychological difficulties experienced by the rescuers

D. The disaster operation

1. The most important prerequisite for good disaster management is that the EMS system must be functioning well on a routine basis. The most important first step is to assess the extent of the disaster and mobilize resources.
2. Prehospital phase
 - a. First responders
 - (1) The major role of law enforcement is to secure the area and assure the safety of emergency workers, victims, and bystanders; in addition, they often establish initial communications.
 - (2) The fire department plays an important role in life safety, rescue and hazard containment, and removing victims from hazardous areas.
 - b. Triage
 - (1) **Definition: an ongoing process of sorting and classifying victims according to treatment priorities; field triage is usually performed by EMS personnel, because physicians and nurses are most useful in hospital settings.**
 - (a) **Primary triage: initial field triage in which classification (usually with a color-coding system) is begun.**
 - (b) **Secondary triage: reexamination, retriage, and further stabilization occurs at the casualty collection area.**
 - (c) **Tertiary triage: reexamination, retriage, and definitive care occurs at the appropriate receiving hospital.**
 - (2) **Categories**
 - (a) **Hopelessly injured (unsalvageable or dead): in the disaster setting, resuscitative efforts and resources should not be expended on any patient in cardiorespiratory arrest or those with injuries deemed unsalvageable.**
 - (b) **Severe (first priority): seriously injured but salvageable patients that require immediate treatment**
 - (c) **Moderate (second priority): seriously injured patients in whom treatment can be delayed without loss of life or limb**
 - (d) **Mild (walking wounded): patients with minor injuries**
 - (3) **Marking triage categories using four colors is the most commonly used system and is based on injury severity and prognosis:**
 - (a) **Black: unsalvageable or dead**
 - (b) **Red: first priority**
 - (c) **Yellow: second priority**
 - (d) **Green: walking wounded**
 - (4) **Triage team: usually consists of experienced EMS personnel. Staff may be drawn from local hospitals, but they must be trained (or experienced) in disaster operations. Triage personnel are under the command of the medical triage officer.**
 - c. Scene control
 - (1) **Overall scene control by fire and rescue personnel to receive and collate information regarding efforts at the scene**
 - (2) **Incident command system established. A command post should be established by the fire personnel at the disaster site (usually uphill and upwind of any hazard).**
 - d. **Communications (the most common problem in a disaster)**

- (1) The disaster plan should provide for a variety of communication systems capable of reaching all responding agencies.
 - (2) Preplanning is required to determine the modes of communication that will be used (landline, cell phone, radio) and to develop protocols for their use.
 - (3) Use of standard language that avoids jargon and "10-codes" facilitates ease of understanding and is mandated under the National Incident Management System.
 - (4) Hospitals should be surveyed to update the availability of critical care and surgical beds.
- e. Miscellaneous considerations
- (1) A support system should be provided for disaster victims and workers to deal with the psychological effects of the disaster.
 - (2) A mechanism for storing the dead must be established.
3. Hospital phase
- a. The Joint Commission requires all hospitals to have a written disaster plan. Disaster drills are required twice yearly.
 - b. Hospitals develop preparedness plans based on a hazard vulnerability analysis. Each hospital has elements of the items below, arranged according to specific risks and needs.
 - c. Emergency Operations Center or hospital command center: An in-house control center that is near but not in the emergency department should be established to monitor the activities of participating hospitals.
 - d. Plan activation: A system of rapid activation must be established that includes a clear delineation of each participant's responsibilities. Additional requirements are the following:
 - (1) A system for calling in necessary personnel
 - (2) Rapid clearing of patients that are currently in the emergency department (and in-house) to make room for the incoming casualties; these patients should be admitted or discharged as appropriate.
 - (3) Establishing a temporary morgue may be necessary.
- e. Treatment areas
- (1) Receiving and triage area: Established near the emergency entrance, tertiary triage is conducted here (primary and secondary triage are generally conducted in the prehospital phase).
 - (2) Critical treatment area: for patients who require the most intensive care; patients stabilized in this area should be placed in an intermediate care area to await surgery or admission.
 - (3) Intermediate treatment area: for patients who are seriously injured but stable
 - (4) Delayed treatment area: for patients whose treatment can be delayed without adverse effect (eg, the walking wounded)
 - (5) Palliative care area: for unsalvageable patients
- f. Documentation
- (1) Patient charts
 - (a) Information should be limited to critical findings and treatments; ideally, the chart should be kept with the patient at all times.
 - (b) Prelabeled kits are preferable to the standard chart system when dealing with unidentified critically ill or injured patients, because the registration process is

cumbersome in this situation. All of the contents are prelabeled with a disaster number. These numbers are acceptable to medical records and laboratory computers and become the patient's "identity" until full registration is possible. Contents of the kit include:

- i. The emergency department record
 - ii. Radiology requests
 - iii. Laboratory slips and tubes
 - iv. Wrist bands
- (2) A separate casualty list should be kept to account for and identify all deceased victims.
- g. Security: extra security is needed very early.
 - h. Waiting areas
 - (1) There should be designated waiting areas away from the treatment areas; media will need an appropriate area, but a separate area must be available for family members only.
 - (2) A hospital public relations officer should act as the public information officer with emergency department personnel, law enforcement, media, and families to provide accurate information to those in the waiting areas.
 - (3) Each waiting area should have adequate telephone access.
4. The National Disaster Medical System (NDMS), created in 1984, is an organization of civilian resources that handles large-scale military or civilian disasters.
 - a. Components of the NDMS
 - (1) Organization of the participating civilian hospitals and health care providers of designated NDMS metropolitan areas.
 - (2) Development of disaster medical assistance teams
 - b. The NDMS is not designated to replace local, state, or regional disaster plans; it is a federal resource available after about 72 hours and is used only in the event of a massive disaster.

EMERGENCY MEDICAL SERVICES: QUICK REVIEW TIPS

Basic Disaster Medical Operations: Triage

Triage is the process of sorting patients according to the severity of injury and the availability of medical resources. In a disaster of mass casualty incident, it is imperative to distinguish between the critically ill and the walking wounded. The first round of triage is usually dedicated to sorting patients into predetermined categories.

Triage tips

- Moribund patients or those in respiratory/cardiac arrest: Patients without spontaneous respirations or without a pulse are generally categorized as "black" or unsalvageable. Triage patients into this category does not preclude palliative treatments such as oxygen or morphine for pain.
- Ambulatory patients: Patients able to follow commands and ambulate without assistance may be tagged as "green" or minor. Patients in this category have sustained minor injuries such as lacerations, abrasions, or sprains.

Reverse triage: Multiple injuries resulting from a lightning strike may benefit from reverse triage. Patients in cardiopulmonary arrest from a lightning strike may benefit from cardiac compressions and resuscitation. This is a reversal of the usual triage process in that a significant amount of resources are dedicated to patients without initial vital signs.

Compliance with the Emergency Medical Treatment and Active Labor Act (EMTALA)

Patients presenting to the emergency department via EMS are essentially requesting a medical screening examination, performed to identify life-threatening conditions and prompt initial stabilizing interventions. Patients transported by EMS have "arrived at the hospital" as soon as the ambulance arrives on hospital property. The lack of prior radio contact or a delay in an orderly transfer of care from EMS to hospital providers does not release the hospital from responsibility under EMTALA. If an emergency department cannot provide services required to stabilize or treat an identified emergency medical condition, then the emergency department is obligated to transfer patients for definitive care. The transfer must take place with resources and personnel sufficient enough to prevent deterioration of the patient's condition en route. If a hospital emergency department is experiencing an internal disaster (eg, flooding, loss of power) or other catastrophic operational condition, it should officially declare "diversion" or "reroute" status so that incoming ambulances are aware of the emergency department's inability to perform medical screening examinations.

NOTES

PROCEDURES AND SKILLS INTEGRAL TO THE PRACTICE OF EMERGENCY MEDICINE

Adult Cardiopulmonary Resuscitation.....	1115
Resuscitative Procedures.....	1116
Noninvasive Positive-Pressure Ventilation	1116
Tracheal Intubation.....	1117
Surgical Airway Management	1122
Venous and Intraosseous Access in Adults	1124
Intraosseous and Central Venous Access in Children and Neonates	1129
Postresuscitative Care	1131
Arterial Catheter Insertion.....	1132
Emergency Department Thoracotomy	1133
Pain Management.....	1136
Acute Pain Management in Adults	1136
Pain Management in Children and Neonates	1138
Procedural Sedation and Analgesia.....	1140
Cutaneous Diagnostic and Therapeutic Procedures	1143
Escharotomy	1143
Incision and Drainage.....	1144
Trephination, Nails	1146
Wound Closure Techniques	1146
Wound Management	1149
Ultrasound.....	1153
Bedside Ultrasound	1153
Focused Assessment with Sonography in Trauma (FAST).....	1154
Aortic Ultrasound	1156
Pelvic Ultrasound	1157
Cardiac Ultrasound.....	1160
Biliary Ultrasound.....	1162
Renal Ultrasound.....	1164
Deep-Vein Thrombosis Ultrasound	1164
Procedural Ultrasound Uses	1165

PROCEDURES AND SKILLS: SELF-ASSESSMENT QUESTIONS

1. A 60-year-old man collapses at work. Optimal citizen's CPR for this patient is:
 - (a) No CPR, just defibrillation with an automatic external defibrillator
 - (b) CPR with a compression/ventilation ratio of 30:2
 - (c) Chest compression only CPR
 - (d) CPR with a compression/ventilation ration of 15:1
2. The goal temperature for post-arrest therapeutic hypothermia is
 - (a) 30 degrees for 12–24 hours
 - (b) 33 degrees for 12–24 hours
 - (c) 30 degrees for 8–12 hours
 - (d) 33 degrees for 8–12 hours
3. Which of the following is true regarding preoxygenation before intubation?
 - (a) Preoxygenation can provide up to 10 minutes of adequate apneic oxygenation.
 - (b) Adequate preoxygenation can be accomplished with 5 minutes of O₂ delivered by nasal cannula at 5 L.
 - (c) Supine position is optimal for preoxygenation.
 - (d) 5 L oxygen via nasal cannula during the apneic period before intubation prolongs the period of adequate oxygenation.
4. Succinylcholine is contraindicated in which of the following?
 - (a) Fresh third-degree burns
 - (b) Acute spinal cord injury
 - (c) Muscular dystrophy
 - (d) Acute crush injury
5. The appropriate size endotracheal tube or tracheostomy tube for placement in an adult patient during cricothyrotomy is:
 - (a) ≤6 mm
 - (b) ≥7 mm
 - (c) 4 mm
 - (d) Depends on size of patient

6. Which of the following is true regarding needle cricothyrotomy?
- (a) A standard 7.0 endotracheal tube adaptor can be attached directly to a 14-gauge angiocatheter.
 - (b) A bag-valve-mask can provide adequate ventilation for adult patients with a needle crich.
 - (c) The initial inspiratory to expiratory ratio with jet ventilation is 1:3.
 - (d) Tubing used must have a Y connector or a hole to allow for exhalation.
 - (e) Options (a) and (d) are correct.
7. Which of the following is true regarding ultrasound guidance for central lines?
- (a) Ultrasound decreases the first attempt success rate.
 - (b) Complication rates between ultrasound-guided and non-ultrasound-guided lines is about the same.
 - (c) Ultrasound increases the number of attempts.
 - (d) Ultrasound is useful for the infraclavicular approach for a subclavian line.
8. Which of the following is true regarding arterial line placement?
- (a) Raynaud phenomenon is not a contraindication to placement of an arterial line in the radial artery.
 - (b) Radial artery pressures are more accurate than femoral in the presence of vasoconstriction.
 - (c) Radial and femoral sites have similar risks of limb ischemia and infection.
 - (d) Anticoagulation is a strict contraindication to arterial line insertion.
9. Which of the following is not a contraindication for procedural sedation in the emergency department?
- (a) ASA of III or higher
 - (b) Snack within the last hour
 - (c) Predicted difficulty with bagging and intubation
 - (d) Procedure expected to last 15 minutes
10. Nonabsorbable sutures retain tensile strength for:
- (a) At least 60 days
 - (b) At least 30 days
 - (c) At least 120 days
 - (d) At least 7 days
11. Which of the following is not true regarding hair removal before suturing?
- (a) It reduces the risk of infection.
 - (b) It can hinder suture removal.
 - (c) Shaving increases the risk of infection 3–9 times compared with clipping.
 - (d) Hair near hairlines and in eyebrows should not be removed.

12. Which of the following is true regarding ultrasound transducers?

- (a) High-frequency transducers are useful for visualizing deep structures.
- (b) Low-frequency transducers penetrate poorly.
- (c) Low-frequency transducers are usually somewhere between 2 and 5 MHz.
- (d) High-frequency transducers have poor resolution.

13. Which of the following is true regarding the FAST examination?

- (a) All unstable patients with any amount of free fluid in the peritoneal space should be taken directly to the operating room.
- (b) The FAST examination is highly accurate for identifying bowel injury.
- (c) The FAST exam is useful in hypotensive medical patients as well as trauma patients.
- (d) Use of the FAST examination reduces mortality and time to the operating room in patients with traumatic pericardial effusions.
- (e) Options (c) and (d) are correct.

ANSWERS

- | | |
|------|-------|
| 1. c | 8. c |
| 2. b | 9. b |
| 3. d | 10. a |
| 4. c | 11. a |
| 5. a | 12. c |
| 6. e | 13. e |
| 7. b | |

Use the pre-chapter multiple choice question worksheet (page xx) to record and determine the percentage of correct answers for this chapter.

I. ADULT CARDIOPULMONARY RESUSCITATION

A. Universal ABCD algorithm

1. Check for responsiveness.
2. Activate emergency response system.
3. Call for defibrillator.

B. Bystander CPR

1. Assess responsiveness, no check for breathing, no pulse check
2. Compression-only CPR shown to increase survival when compared with conventional CPR and no CPR

C. Healthcare provider CPR

1. ABCD
 - a. Airway: open airway
 - b. Breathing: look, listen, feel
 - c. Circulation: check pulse, start CPR within 10 sec
 - d. Defibrillator: attach monitor/defibrillator
2. Analyze rhythm: shock if ventricular fibrillation/pulseless ventricular tachycardia
3. CPR for 2 min at a rate of at least 100/min; 30 compressions/2 breaths until definitive airway established
4. Secondary ABCD
 - a. Secure airway
 - b. Confirm placement and adequacy of ventilation
 - c. Intravenous/intraosseous access, medications, monitor and determine rhythm
 - d. Differential diagnosis
5. Treat reversible causes

Table 48: Potentially Reversible Causes of Cardiac Arrest

Hypovolemia/hemorrhage (IV fluids, blood)	Tablets/Trauma – overdose (naloxone)
Hypoxia (O ₂ , ventilate)	Tamponade (pericardiocentesis)
Hydrogen/acidosis (ventilate, possibly bicarbonate)	Tension pneumothorax (needle decompress)
Hyperkalemia (calcium, insulin/D50)	Thrombosis – acute coronary syndrome (tissue plasminogen activator versus percutaneous coronary angioplasty [PTCA] if resuscitated)
Hypothermia (warm)	Thrombosis – pulmonary embolism (tissue plasminogen activator)

6. Continue CPR, check pulse every 2 minutes.
7. If ventricular fibrillation/ventricular tachycardia
 - a. Defibrillate every 2 minutes
 - b. Pads preferred over paddles (improved contact and safety)
 - c. Monophasic defibrillator: 360 joules for all shocks
 - d. Biphasic: follow manufacturer's recommendation; usually 150 joules for first shock, then 200 joules

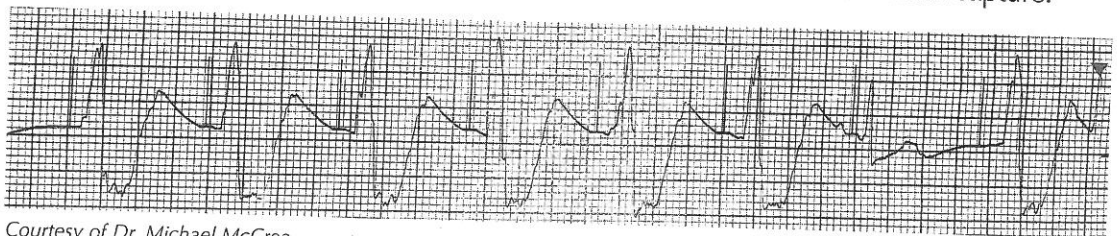
8. Medications: introduce after first 2 minutes of CPR.
 - a. Epinephrine 1 mg every 3 minutes
 - b. Amiodarone 300 mg once
 - c. Lidocaine 1–1.5 mg/kg, then 0.5 mg/kg every 5 minutes (maximum 3 mg/kg)
 - d. Magnesium 1–2 g IV push (consider if concern for torsades de pointes)
 - e. Calcium chloride 1 ampule (consider if concern for hyperkalemia)
 - f. Bicarbonate (consider in prolonged arrest)
 - g. Atropine 1 mg every 5 minutes in for asystole/pulseless electrical activity <60, up to 3 mg
9. Return of spontaneous circulation (ROSC)
 - a. Therapeutic hypothermia
 - b. PTCA

D. Synchronized cardioversion

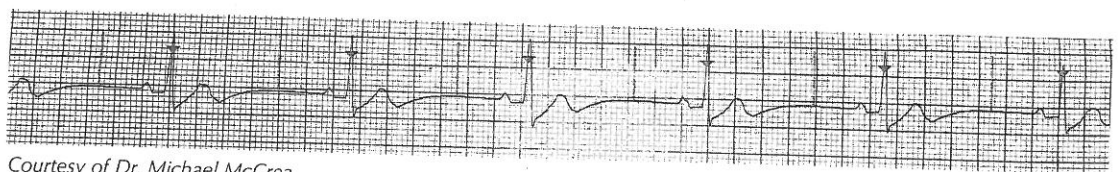
1. Indication: unstable narrow or wide complex tachycardia
2. Technique: place defibrillator pads; set defibrillator to SYNC, charge, and defibrillate.
3. Re-SYNC if second cardioversion required.

E. Pacing

1. Indication: symptomatic bradycardia/block, consider in asystole
2. Technique: place defibrillator pads. Turn PACER on. Set HEART rate (70–80). Set initial pacer CURRENT (MILLIAMPS). Increase until capture is evidenced by pacer spikes followed immediately by a QRS complex. Check pulse to ensure mechanical capture.



Courtesy of Dr. Michael McCrea



Courtesy of Dr. Michael McCrea

3. Move immediately to definitive care, transvenous pacing.

II. RESUSCITATION PROCEDURES

A. Noninvasive positive-pressure ventilation

1. Indicated for respiratory distress due to COPD and cardiogenic pulmonary edema.
2. Both continuous positive-airway pressure (CPAP) and bi-level positive-airway pressure (BiPAP) are effective in reducing rates of intubation/intubation complications.

3. Contraindications: altered mental status, vomiting or excessive secretions, inability to wear/tolerate mask
4. Cautions: abdominal distension, oral secretions, auto-positive end-expiratory pressure (PEEP) in COPD, reassess frequently, intubate if not improved
5. Pre-hospital CPAP
 - a. Decreases emergency department intubations and overall mortality
 - b. Class IIa intervention for cardiogenic pulmonary edema
6. CHF in emergency department
 - a. No advantage of BiPAP over CPAP
 - b. Improves heart rate, blood pressure, O_2 saturation, PaO_2 , A-a gradient
 - c. Decreases mortality
7. COPD in emergency department
 - a. Only advantageous in severe distress
 - b. Decreases hypoxia, acidosis, hypercapnia within 1 hour
 - c. Reduces length of stay, intubation rate, mortality
8. Settings
 - a. May use full mask or nasal pillows
 - b. CPAP: 5 cm/ H_2O , titrate up by 1–2 cm/ H_2O
 - c. BiPAP – inspiratory positive-airway pressure (IPAP) 8–10, expiratory positive-airway pressure (EPAP) 4–5
 - (1) Most studies show effective range IPAP 14–20, EPAP 5–8
 - (2) Increase EPAP by 1–2, maintain EPAP:IPAP of about 2.5:1
 - d. Oxygen bleed-in to maintain O_2 saturation >90%

B. Tracheal intubation

1. Indications
 - a. Inability to protect airway
 - b. Hypoxia not corrected with oxygen
 - c. Hypercarbia
 - d. Hypoventilation
 - e. Respiratory failure or impending failure
2. Routes
 - a. Nasotracheal
 - (1) Infrequently used because more morbidity than endotracheal intubation but may be indicated for spontaneously breathing patients with angioedema of tongue, clenched teeth
 - (2) Special nasal intubation that allows flexion of the tip highly recommended
 - (3) Insert lubricated tube (no stylet) through largest nare that has been prepared with topical vasoconstrictor.
 - (4) Rotate tube medially, listen for breath through tube, advance tube during initiation of inspiration using breath sounds to guide tube.
 - (5) Advance to 28 cm at nare for men, 26 cm for women
 - (6) May be used in conjunction with fiberoptic scope for patients at high risk of failed airway

- b. Orotracheal
 - (1) Standard direct laryngoscopy versus video-assisted
 - (2) Tube size: 8 for men, 7–7.5 for women, $(\text{age} + 16)/4$ for children
- 3. **Rapid-sequence intubation**
 - a. **Simultaneous administration of induction and paralytic agent**
 - b. **Method of choice in emergency department intubations: highest success rates, fewest complications**
 - c. **Exceptions**
 - (1) Cardiac/respiratory arrest, patient with no gag reflex
 - (2) Spontaneously breathing patient with high risk of inability to ventilate or intubate (see below)
 - d. **Airway evaluation**
 - (1) Used to predict difficulty with bag-valve-mask ventilation and/or intubation
 - (2) Indicated except in cases of crash airway (respiratory arrest)
 - (3) **Predictors of difficulty with ventilation: presence of ≥ 2 factors predictive of inability to ventilate effectively**
 - Obesity (also pregnancy): difficult to achieve adequate tidal volume
 - Beard: difficult seal
 - Elderly (over 55)
 - Snorers: airway obstruction
 - Edentulous: difficult seal

Table 49: LEMON Rule for Prediction of Difficult Intubation

	Predictor of Difficult Intubation
Look externally	Sunken cheeks Edentulous Protruding incisors Micrognathia
Evaluate 3-3-2 rule	Mouth opening <3 fingers Hyoid–chin distance <3 fingers Thyroid cartilage to mouth floor <2 fingers
Mallampati Score	III or IV
Obstruction	Oral obstruction (abscess, trauma)
Neck mobility	Limited range of motion of neck (arthritis, fixation, fracture)

- e. **Steps**
 - (1) **Preparation**
 - (a) Standard intubation equipment open and tested
 - (b) Oxygen and suction on
 - (c) Capnography or other confirmation tool at hand
 - (d) Personnel present
 - (e) Medications drawn up
 - (f) Rescue airway and surgical airway at bedside

- (2) Preoxygenation
 - (a) Displaces nitrogen with oxygen in alveolar space creating oxygen reservoir
 - (b) Provides up to 8 minutes of adequate oxygenation during apnea
 - (c) Adequate oxygenation time decreased in pregnancy, obesity, pulmonary disease, hyperdynamic states (sepsis, trauma), extremes of age
- (3) Technique
 - (a) 3 minutes high-flow oxygen by nonrebreathing mask or
 - (b) 8 vital capacity breaths with nonrebreathing mask
 - (c) Elevation of head of bed 25° shown to be effective
 - (d) 5 L O₂ via nasal cannula during apneic period before intubation shown to extend period of adequate oxygenation
- f. Patient position
 - (1) Slight flexion of neck with extension of atlanto-occipital joint: sniffing position
 - (a) Aligns oropharyngeal-laryngeal axis
 - (b) Improper position and improperly prepared equipment are the two most common causes of intubation failure.
 - (2) Morbidly obese patients: ramped position
 - (a) Head and shoulder elevated above chest
 - (b) External auditory canal parallel to sternal notch
 - (c) May require multiple folded blankets under head and neck
- g. Pretreatment
 - (1) Theoretically blunts physiologic reactions to laryngoscopy and intubation but scant evidence
 - (2) Agents
 - (a) Lidocaine 1–1.5 mg/kg: recommended for increased intracranial pressure (ICP) and bronchospasm but no evidence of improved outcomes
 - (b) Fentanyl 3 mcg/kg: opiate, used to minimize sympathetic response to intubation, may be useful in patients with increased ICP, dissection, aneurysm but may cause chest rigidity and respiratory depression
 - (c) Atropine 0.02 mg/kg (minimum dose 0.10 mg): recommended in children with bradycardia and in adults (0.01 mg/kg) with bradycardia after repeat succinylcholine
 - (3) Pretreatment with nondepolarizing agents before administration of succinylcholine is no longer recommended.
- h. Potent sedative and paralysis
 - (1) Administer sedative before neuromuscular blocking agent
 - (2) All with rapid onset (≤ 2 minutes), duration 10–15 minutes

Table 50: Sedative Agents

Agent	Dosage	Benefits	Cautions
Etomidate	0.3 mg/kg	Little hemodynamic effect Decreases ICP	Myoclonus Vomiting No analgesia Decreased cortisol release*
Propofol	1.5–3 mg/kg	Bronchodilation Decreases ICP Anticonvulsant	Hypotension No analgesia Apnea
Ketamine	1–2 mg/kg	Increased blood pressure Bronchodilation	Use in increased ICP controversial
Midazolam	0.2–0.3 mg/kg	Anticonvulsant Potent amnestic	Hypotension Frequently underdosed

* Etomidate associated with transient suppression of adrenal function but no clear change in mortality; no study to date powered to detect effects on hospital or ICU length of stay, ventilator days, or mortality.

- (3) Ketamine 2 mg/kg has been shown to be safe alternative to etomidate in rapid-sequence intubation of critically ill patients; may be considered in sepsis.
- (4) For obese patients, etomidate should be dosed based on lean body weight, and midazolam initial bolus dosed on actual body weight with maintenance dosing based on ideal body weight.
- (5) Propofol should be dosed based on lean body weight.
- i. Neuromuscular blocking agents
 - (1) Succinylcholine 1.5 mg/kg (based on actual body weight)
 - (a) Drug of choice for rapid-sequence intubation because of its rapid onset (45–60 seconds) and short duration of action (5–9 minutes) unless contraindicated.
 - (b) Potential complications
 - i. Hyperkalemia in:
 - Burns >5 days old
 - Denervation injury >5 days old
 - Severe crush injury >5 days old
 - Severe infection >5 days old
 - Known hyperkalemia
 - Preexisting myopathies
 - ii. Fasciculations
 - iii. Masseter spasm
 - iv. Malignant hyperthermia
 - v. Bradycardia
 - vi. May increase intraocular pressure and ICP, although recent studies refute this

Table 51: Nondepolarizing Agents

Agent	Dosage	Onset (min)	Duration (min)	Comments/Cautions
Rocuronium	1 mg/kg	1–3	30–45	First choice alternative to succinylcholine
Vecuronium	0.10 mg/kg	2–4	25–40	Longer duration in hepatorenal failure
Atacuronium	0.5 mg/kg	2–3	25–45	Hypotension, bronchospasm

(2) Succinylcholine is dosed based on total body weight in obese patients.

(3) Rocuronium is dosed based on actual body weight in obese patients.

4. Placement with confirmation

a. Technique

- (1) Place straight or curved blade to right side of mouth, sweep tongue to the left.
 - (a) Straight blade: lift epiglottis
 - (b) Curved blade: insert into vallecula and lift up and away
- (2) Visualization shown to be improved with tracheal manipulation by intubator with position then maintained by assistant
- (3) Routine cricoid pressure is no longer recommended.
- (4) If unable to visualize larynx, withdraw and try secondary method.
- (5) Gently insert tube on right side of mouth, rotate through cords.
- (6) Difficulties with passing tube usually due to too large tube size, cricoid pressure, improper stylet bend (35° considered optimal)
- (7) Withdraw blade, inflate cuff <40 cm/H₂O to avoid mucosal ischemia

b. Confirmation

- (1) Visualization of tube through cords is primary confirmation
- (2) Lung sounds assist in detecting right main-stem bronchus (RMS) intubation.
- (3) Secondary confirmation
 - (a) Continuous end-tidal CO₂ nearly 100% accurate in confirmation
 - (b) Colorimetric end-tidal CO₂
 - i. Give six breaths to ensure not false positive
 - ii. May produce false negative in low-flow states
 - (c) Syringe aspiration
 - i. 30-mL syringe attached to endotracheal tube, withdraw.
 - ii. If in trachea, air is easily withdrawn.
- (4) Chest radiograph to confirm absence of RMS intubation but not a reliable means to confirm endotracheal placement

5. Postintubation care

- a. Secure tube in place without decreasing venous return
- b. Place orogastric or nasogastric tube
- c. Maintain adequate sedation/analgesia to prevent agitation/extubation
- d. Usual initial ventilator settings

- (1) Mode: assist control (A/C)
 - (2) FiO_2 : 100%, decrease to maintain PaO_2 60%–90%
 - (3) Tidal volume 10 mL/kg (use ideal body weight in obese patients)
 - (4) Respiratory rate: 12 breaths per minute, increase in acidosis
 - (5) Inspirations:expirations 1:2; increase expiratory duration for obstructive lung disease
 - (6) PEEP: 5 cm H_2O
6. Troubleshooting
 - a. Disconnect from vent and use bag-valve-mask
 - b. Suction tube
 - c. Evaluate for pneumothorax/auto PEEP
 - d. Reevaluate ventilator settings
 7. Difficult airway tools
 - a. Gum elastic bougie
 - (1) Flexible, plastic rod with angled tip
 - (2) Used with direct laryngoscopy when only arytenoid cartilage visible
 - (a) Thread endotracheal tube over bougie.
 - (b) Insert bougie into trachea using direct laryngoscopy.
 - (c) Confirm placement by advancing bougie to carina and feeling firm tracheal rings when advancing, retracting bougie.
 - b. Video-assisted intubation
 - (1) Shown to improve visualization of cords with minimal neck movement
 - (2) Limited by secretions or blood in airway
 - (3) Special stylet may be required
 - (4) Insert blade in midline of tongue, advance along midline, insert into vallecula similar to curved blade.
 - c. Fiberoptic
 - (1) Requires training/practice, set up time, and compliant, spontaneously breathing patient
 - (2) Nasal preferred to oral route: topical anesthetic and vasoconstrictor keys to success
 - (3) Requires ≥ 7.5 endotracheal tube
 - (4) Thread tube over fiberoptic scope (adapter removed)
 - (5) Pass fiberoptic scope through cords and advance tube
- C. Surgical airway management
1. Surgical cricothyrotomy
 - a. **Indication: failed airway, ie, can't ventilate, can't intubate**
 - b. **Minimum equipment required**
 - (1) Scalpel (10 or 11 blade)
 - (2) 6 mm or less endotracheal or tracheostomy tube
 - (3) Tape or ties to secure device
 - (4) Bag-valve-mask with oxygen source
 - c. **Technique**
 - (1) **Prepare site**

- (2) Right-handed operator stands at right side of patient
 - (3) Palpate anterior trachea from sternum superiorly
 - (4) Identify cricoid ring –most inferior palpable prominence
 - (5) Identify cricoid notch immediately superior to ring
 - (6) Grasp laryngeal cartilage in nondominant hand
 - (7) Make vertical incision 1–2 inches over cricoid notch, cutting through skin and subcutaneous tissue.
 - (8) Make horizontal stab incision through cricothyroid membrane
 - (9) Place back end of scalpel into incision and rotate to dilate (or use dilator or Kelly clamp or dilator)
 - (10) Insert endotracheal or tracheostomy tube. If using endotracheal tube, insert only 2–3 cm to avoid RMS intubation, and cut off top of tube to minimize amount of tube external to neck.
 - (11) Key: once the membrane is cut, keep the scalpel, dilator or tracheal hook in the hole at all times.
- d. Alternative techniques
- (1) Once scalpel incision has been made, insert bougie into hole, thread endotracheal tube over bougie, inflate and secure.
 - (2) Some operators prefer to make a single horizontal incision through the skin, subcutaneous tissue and cricoid membrane. This requires that the cricoid membrane is confidently identified before making the incision.
2. Needle cricothyrotomy
- a. Recommended instead of surgical cricothyrotomy in patients ≤ 8 -10 years old
 - b. May be used in adults but limits on ventilation mandate replacement within 15–20 minutes
 - c. Equipment
 - (1) 3-mL syringe with 12- or 14-gauge angiocatheter attached
 - (2) Wall oxygen source with 15 L/min flow (bag-valve-mask does not provide adequate ventilation)
 - (3) Adaptor from 7.0 endotracheal tube
 - (4) Tubing with side hole or Y connector
 - d. Technique
 - (1) Identify cricothyroid membrane.
 - (2) Insert needle, at 90° angle to skin while aspirating; membrane is punctured when air enters into syringe.
 - (3) Change angle to 45° (directed inferiorly).
 - (4) Advance catheter off needle, remove needle.
 - (5) Attach endotracheal tube adaptor to catheter, or reattach syringe barrel (plunger removed) to catheter and attach 7 mm endotracheal tube adaptor to syringe.
 - (6) Connect to tubing.
 - (7) Jet ventilation unit preferable to unregulated wall-source oxygen.
 - (8) Initial I:E ratio should be 1:4 (may need to increase to 1:10 or more to ensure adequate exhalation).
 - (9) Immediate preparation for alternative airway must be made.

D. Venous and intraosseous access in adults

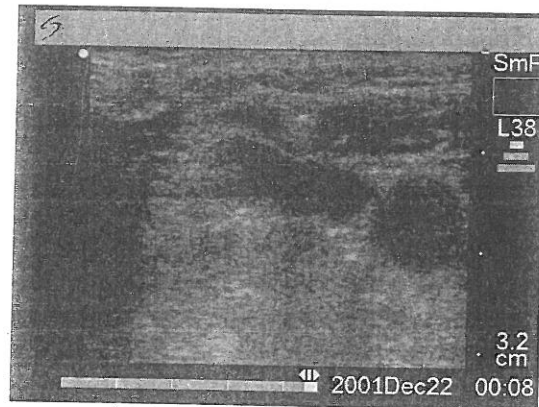
1. Necessary for administration of fluids, medications, blood products
2. Route of access determined by:
 - a. Availability of access
 - b. Infusion rate requirement: maximal rate determined by Poiseuille's law
 - (1) Most important factor is internal catheter diameter
 - (2) Also important are catheter length and pressure gradient
 - c. Types of medications to be administered
 - d. Need for invasive monitoring
3. Peripheral access
 - a. First choice
 - b. Large diameter, short catheter preferred
 - c. Most common sites: hand veins, veins of antecubital fossa
 - d. Lower-extremity veins often require cut-down
 - e. Relative contraindications: sclerosing agents, chemotherapeutic agents, concentrated electrolyte or glucose solutions
 - f. Extremity with A-V fistula/graft
 - g. Lower-extremity veins in diabetic patients
 - h. Distal to site of injury or infection
 - i. Distal to site of vascular disruption
 - j. Ultrasound guidance: useful in obesity, shock states, end-stage renal disease, intravenous drug use, burn scars, history of multiple IVs (sickle cell patients)
 - k. Most common sites are basilic and cephalic veins
 - (1) High rate of success, few complications, avoid need for central access
 - (2) May require longer catheter
 - l. Complications
 - (1) Extravasation of fluids
 - (2) Superficial thrombophlebitis (2%–12%)
 - (3) Tissue necrosis
 - (4) Cellulitis
 - (5) Hematoma
4. **Central access: general technique (Seldinger)**
 - a. Obtain consent.
 - b. Identify vessel by landmarks or with ultrasound.
 - c. Sterile prep/gown/drape.
 - d. Open kit and prepare catheter, guidewire.
 - e. Anesthetize area in conscious patients.
 - f. Insert 18-gauge needle attached through skin in direction of vein.
 - g. Aspirate while inserting.
 - h. Withdraw while aspirating if needed, and reposition needle.
 - i. Once blood is aspirated, remove syringe from needle, watch for continued blood flow.
 - j. Insert wire through needle, advance ~10 cm.

- k. *Keep firm grip on wire.*
 - l. Remove needle over wire.
 - m. Make skin nick with scalpel.
 - n. Dilate subcutaneous tissue with dilator.
 - o. Remove dilator.
 - p. Insert catheter over wire keeping firm grasp on proximal end of wire until free end is through distal port.
 - q. Remove wire.
 - r. Aspirate blood through all ports.
 - s. Secure line, dress.
 - t. Obtain chest radiograph for subclavian and internal jugular lines.
 - (1) Tip should be in superior vena cava
 - (2) Look for pneumothorax (but may have delayed presentation)
5. Ultrasound guidance
- a. Increases rate of success on initial attempt
 - b. Decreases number of attempts
 - c. Rate of complications similar to that of non-ultrasound guided
6. Complications
- a. General
 - (1) Arterial puncture
 - (2) Hematoma
 - (3) Infection
 - (4) Thrombosis
 - b. Internal jugular and subclavian
 - (1) Tracheal injury
 - (2) Chylothorax (thoracic duct injury on left side)
 - (3) Hemothorax (great vessel or right atrial injury)
 - (4) Pneumothorax
7. Approaches
- a. Internal jugular
 - (1) Right internal jugular preferred
 - (2) Shorter, more direct route to superior vena cava
 - (3) Avoids injury to thoracic duct on left
 - b. Traditional approaches
 - (1) Central
 - (a) Identify apex of triangle created by clavicle and sternal/clavicular heads of sternocleidomastoid muscle.
 - (b) Insert needle 1 cm inferior to apex at 30° angle, directed toward ipsilateral nipple.



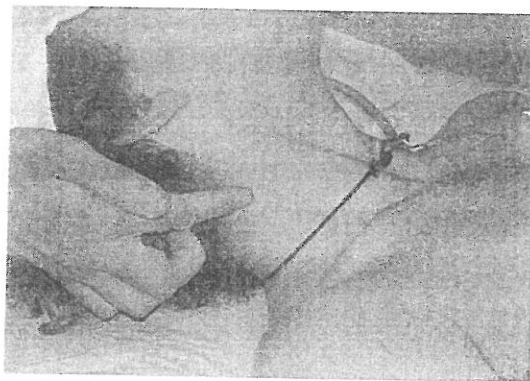
Courtesy of Jessica Resnick, MD, RDMS, FACEP

(c) Advance 1–3 cm while aspirating.



(2) Posterior

(a) Identify clavicular portion of sternocleidomastoid muscle.



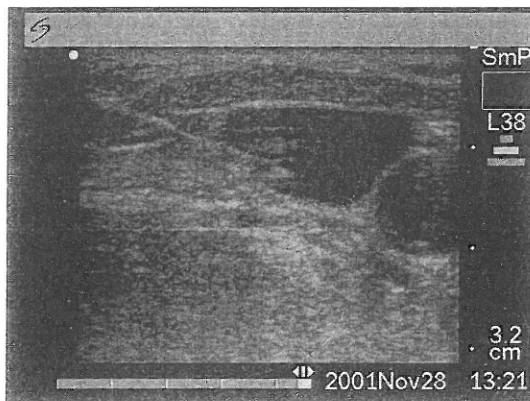
Courtesy of Jessica Resnick, MD, RDMS, FACEP

- (b) Insert needle under muscle belly 1/3 distance from clavicle to mastoid process.

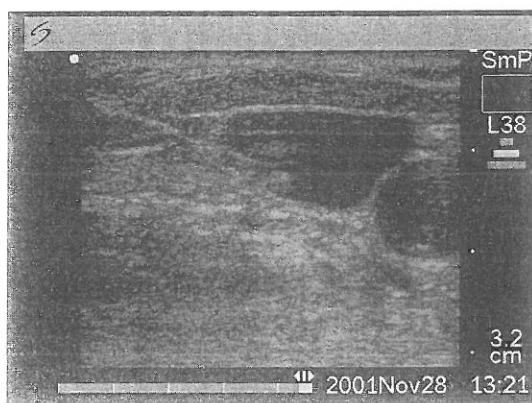


Courtesy of Jessica Resnick, MD, RDMS, FACEP

- (c) Direct needle toward sternal notch.
(d) Advance 3–5 cm while aspirating.



- (3) Anterior
- Palpate carotid artery with nondominant hand.
 - Insert needle lateral to carotid artery at center of sternal portion of sternocleidomastoid muscle.
 - Direct needle toward ipsilateral nipple.
 - Advance 3–5 cm while aspirating.
- (4) Ultrasound guided
- Place ultrasound transducer in transverse plane superior to clavicle.
 - Identify internal jugular, carotid artery, thyroid cartilage.
 - Compress vein to exclude thrombosis and differentiate from artery.
 - Enter vein in center of long face of transducer (traditional central approach), or enter vein in center of short face of transducer and follow needle into vein (follow course of traditional posterior approach).



- c. Subclavian: allows neck mobility, good choice for long-term use but increased risk of pneumothorax over internal jugular cannulation, check for pneumothorax if failure on one side before attempting on contralateral side.
 - (1) Infraclavicular approach
 - (a) Trendelenburg, head neutral
 - (b) Towel roll under upper mid back to elevate clavicle
 - (c) Place index finger of nondominant hand at midpoint of clavicle, thumb at sternal notch.
 - (d) Insert needle at junction of middle and medial segments with bevel directed inferiorly so wire does not go up into internal jugular.
 - (e) Needle should be just under the clavicle: walk needle down clavicle and then advance.
 - (f) Advance needle 3–5 cm while aspirating.
 - (2) Supraclavicular approach
 - (a) Advantages over infraclavicular – lower failure rate, lower rate of catheter malposition, less interference with CPR, can be performed in upright position
 - (b) Technique
 - i. Trendelenburg, head neutral
 - ii. Needle entry 1 cm lateral to clavicular head of sternocleidomastoid muscle, 1 cm posterior to clavicle
 - iii. Angle 10° from horizontal to skin
 - iv. Bevel angled medially
 - v. Aim to contralateral nipple
 - vi. Advance needle 2–3 cm
 - (3) Ultrasound guidance
 - (a) Not useful for infraclavicular
 - (b) Supraclavicular
 - i. Place transducer just superior to medial aspect of clavicle in transverse plane
 - ii. Needle entry same as traditional supraclavicular approach
- d. Femoral
 - (1) Most accessible site for central access during cardiac arrest
 - (2) Less desirable site because of higher rates of infection, thrombosis

(3) Technique

- (a) Patient supine, in reverse Trendelenberg
- (b) Leg slightly abducted and externally rotated
- (c) Palpate femoral artery 1–2 cm below inguinal ligament
- (d) Vein is medial to artery
- (e) Insert needle at 45° angle: *must* be below inguinal ligament

(4) Ultrasound guidance

- (a) Place transducer in transverse orientation 2–3 cm inferior to inguinal ligament
- (b) Identify vein medial to artery: larger and more easily compressed
- (c) Center vein in middle of transducer
- (d) Insert needle as in traditional approach

8. Intraosseous (IO) access

- a. Indication: patient of any age when venous access cannot be rapidly established in presence of circulatory collapse
- b. Contraindications
 - (1) Proximal ipsilateral fracture or vascular injury (use other side)
 - (2) Severe osteoporosis or osteogenesis imperfecta
- c. Access sites
 - (1) Proximal humerus
 - (2) Medial malleolus
 - (3) Distal femur
 - (4) Sternum
 - (5) Tibia not recommended: thick and difficult to penetrate
- d. Technique
 - (1) Use standard bone marrow or specialized infusion needle.
 - (2) Direct needle perpendicular to bone.
 - (3) Twist needle maintaining constant pressure.
 - (4) Remove stylet after pop through bone is felt.
 - (5) Confirm with aspiration.
 - (6) Commercial gun or drill may be used in place of manual technique.
- e. Infusion/medications
 - (1) Any fluid, blood product, medication appropriate for parenteral administration can be given IO.
 - (2) RSI medications: slightly slower onset
 - (3) Infusion rate limited by bone marrow absorption: improved with pressure bag.

E. Intraosseous and central venous access in children and neonates**1. Intraosseous access**

- a. Indicated for emergency vascular access
- b. Demonstrated more rapid access than peripheral route even with manual IO insertion
- c. Serious complications (osteomyelitis, fracture, growth plate injury, air/fat emboli, compartment syndrome) <1% compared with 3.4% for central venous catheterization

- d. Sites
 - (1) Proximal tibia, anteroomedial surface: 2 cm distal to tibial tuberosity
 - (2) Proximal humerus: recommended for patients ≥ 5 years old
 - (3) Distal tibia
 - (4) Proximal femur (not FDA approved)
- e. Technique
 - (1) Similar to that for adults (see above)
 - (2) Use pediatric size needle.
 - (3) Commercial gun or drill may be used.
- 2. Central venous access
 - a. Indications for emergency department pediatric central access
 - (1) Inability to obtain peripheral access
 - (2) Invasive monitoring
 - (3) Caustic/hypertonic solutions
 - (4) Need for long-term access
 - (5) Transvenous pacing
 - b. Complications
 - (1) Insertion of >6 French catheters in children <1 year old, <10 kg associated with higher complication rate.
 - (2) Use 5 French catheter in infants, 3 French in neonates.
 - (3) See adult section for additional complications.
 - c. Technique: similar to that for adults except as below
 - d. Subclavian
 - (1) Head in neutral position, but no towel roll under back.
 - (2) Pneumothorax more likely as lung rises above clavicle.
 - (3) Right side preferred, as in adults; also, right lung dome is lower than left internal jugular.
 - (4) Head position 45° from midline
 - (5) Ultrasound guidance recommended
 - (6) Combination of Trendelenburg, Valsalva, and liver compression maximizes vein size in children (negligible in infants).
 - e. Femoral
 - (1) Advantages
 - (a) Easier in uncooperative child
 - (b) Fewer immediate complications
 - (c) Does not interfere with CPR
 - (2) Disadvantage: possibly higher infection rate
 - (3) Technique: similar to that for adults
 - (a) Artery more likely to overlap vein so ultrasound guidance recommended
 - (b) Line may be misdirected into hepatic vein; seen as curled line on radiograph
 - f. Neonatal umbilical vein cannulation
 - (1) Usually limited to first week of life

- (2) Anatomy
 - (a) Two small arteries, thick walled
 - (b) One larger vein, thin walled: usually at 12:00 position, continuous with portal vein
- (3) Technique
 - (a) Use 4–5 cm 5.0 French catheter for term neonates, 3.5 French for preterm.
 - (b) Flush catheter with heparinized solution and attach to syringe with 3-way stopcock.
 - (c) Prepare lower abdomen. Use sterile technique.
 - (d) Place purse-string suture or umbilical tie loosely around abdominal aspect of umbilical stump.
 - (e) Cut the stump with scalpel ~2 cm from abdominal wall.
 - (f) Clear thrombus with forceps, dilate vein if needed.
 - (g) Gently insert catheter, direct toward right shoulder.
 - (h) Check every centimeter for blood return by opening stopcock and gently withdrawing on syringe.
 - (i) If met with resistance, loosen suture slightly
 - (j) Advance catheter 1–2 cm beyond point of good blood return.
 - (k) Tighten and secure suture or tie.
 - (l) Monitor for signs of bowel or vessel perforation.

F. Postresuscitative care

1. Postresuscitation syndrome

- a. Results from as little as 5 minutes of transient ischemia
- b. Cascade of vascular, chemical, molecular events leading to permanent injury
- c. Occurs in all organs but brain is most sensitive
- d. Cerebral effects
 - (1) Edema
 - (2) Loss of blood-brain barrier
 - (3) Alterations in blood flow
 - (4) Mitochondrial damage
 - (5) Enzyme release
- e. Systemic effects
 - (1) Hypotension
 - (2) Hypoxia
 - (3) Hypercarbia
 - (4) Electrolyte imbalance
 - (5) Disseminated intravascular coagulation
 - (6) Bacteria translocation from intestines to blood

2. Postresuscitation care

- a. First priority: restore circulation, ventilation, oxygenation
- b. Second priority: prevent rapid rewarming and fever

3. Therapeutic hypothermia

a. Evidence

- (1) Two studies demonstrated improved outcomes in patients after ventricular fibrillation arrest who were cooled and maintained in mild hypothermia for 12–24 hours after ROSC.
- (2) Further studies demonstrated benefit in other arrest situations, providing ROSC attained in <25 min
- (3) Neonates with birth asphyxia shown to have decreased mortality and incidence of cerebral palsy
- (4) Limited information on use in pediatric population

b. Indications

- (1) Ventricular fibrillation/ventricular tachycardia arrest or near drowning ROSC <60 minutes (consider in pulseless electrical activity with ROSC <30 minutes)
- (2) Comatose/intubated, Glasgow Coma Score ≤ 9 , temperature $>34^{\circ}$
- (3) Mean arterial pressure >80 mmHg (if low, correct with IV fluids and dopamine)
- (4) No contraindications (do-not-resuscitate, sepsis, cancer with brain metastases, active bleeding, advanced dementia)

c. Technique (induced cooling to eliminate deficits = ICED)

- (1) Cooling: goal is 33°
 - (a) Cold IV fluids (lactated Ringer's) $\times 2$ L
 - (b) Ice packs/ice soaked sheets./commercial cooling device
- (2) ECG: if evolving MI, prepare for PTCA while performing ICED.
- (3) Medications
 - (a) Sedation/seizure prevention: fentanyl or midazolam
 - (b) Prevention of shivering: paralysis with vecuronium
 - (c) ASA per rectum if no contraindications
- (4) Additional components
 - (a) Maintain end-tidal CO_2 35–40 mmHg
 - (b) Treat blood glucose >150 mg/dL with regular insulin IV
 - (c) Transfuse if hemoglobin <10 mg/dL
 - (d) Central line if indicated

G. Arterial catheter insertion

1. Indications

- a. Refractory shock
- b. Management of vasopressor therapy
- c. Frequent arterial blood gas sampling
- d. Calculation of CO and pulse pressure variations

2. Contraindications

a. Strict

- (1) Inadequate circulation to extremity
- (2) Raynaud syndrome
- (3) Buerger disease
- (4) Full-thickness burns

- b. Relative
 - (1) Previous surgery/cut down in area
 - (2) Anticoagulation, coagulopathy
 - (3) Inadequate collateral flow
 - (4) Partial thickness burns
- 3. Sites
 - a. Radial: most common
 - b. Femoral
 - (1) Easier to access in emergencies
 - (2) More accurate in presence of vasoconstriction
 - (3) Similar risk of infection/limb ischemia
 - (4) Complications: retroperitoneal hematoma and pseudoaneurysm
 - c. Other (rarely used): axillary, brachial, ulnar, dorsalis pedis, posterior tibialis, temporal
- 4. Technique
 - a. Sterile prep
 - b. Radial artery – place towel under dorsal wrist/maintain wrist in extension
 - c. Identify pulse
 - (1) Palpation with index and middle finger of nondominant hand
 - (2) Ultrasound useful in hypotensive/obese patients
 - d. Insert catheter (use arterial catheter).
 - (1) Bevel up
 - (2) Directly over the needle or Seldinger technique
 - e. Attach tubing (heparinized).
 - f. Connect to pressure transducer.
 - (1) Square wave flush test to ensure appropriate dampening.
 - (2) 1.5–2 oscillations after flush indicates no air in line.
- 5. Complications
 - a. Limb ischemia (0.1%): Allen test (simultaneous compression of radial/ulnar arteries with release of ulnar artery pressure) not reliable for predicting hand ischemia
 - b. Temporary occlusion (20% in radial artery, 1.5% femoral)
 - c. Hematoma (14% radial, 6% femoral)
 - d. Infection
 - e. Pseudoaneurysm

H. Emergency department thoracotomy

- 1. Patients with penetrating chest trauma who arrest in the field or in the emergency department are most likely to benefit (limited evidence for penetrating abdominal injuries). Patients with blunt trauma, prolonged arrest, delayed transport are not likely to benefit.
- 2. Indications: penetrating
 - a. At least one of the following signs of life on arrival at the emergency department:
 - (1) Blood pressure
 - (2) Pulse

- (3) Cardiac rhythm
- (4) Respiratory effort
- (5) Echo cardiac activity
- b. If no signs of life on arrival but
 - (1) Echo shows tamponade *or*
 - (2) Signs of life at scene and paramedic CPR <10 minutes
- 3. Indications: blunt
 - a. At least one of the above signs of life on arrival at the emergency department *or*
 - b. Echocardiographic evidence of tamponade with paramedic CPR <10 minutes *or*
 - c. Cardiac activity after initiation of airway, IV fluids, and needle chest decompression
- 4. Indications: pediatric—little evidence, recommendation is to follow adult guidelines.
- 5. Technique
 - a. Preparation
 - (1) Personal protective equipment
 - (2) Airway secure: consider purposeful RMS intubation to prevent left lung inflation from hampering procedure
 - (3) Nasogastric tube time permitting
 - (4) Analgesia, paralytics available if patient regains pulse
 - (5) Prep left chest if reasonable
 - (6) Patient's left arm over head, towels under left scapula
 - b. Incision/entry
 - (1) Anterolateral chest at fourth or fifth rib space (inframammary fold)
 - (2) 20 blade scalpel
 - (3) Incise from right side of sternum past left posterior axillary line, through skin, subcutaneous tissue, and superficial muscle.
 - (4) Cut intercostal muscles with scissors.
 - (a) Cut above rib to avoid vessels/nerves
 - (b) Scissors reduce laceration risk to operator
 - (5) Stop ventilation: cut through pleura
 - (6) Place rib spreader: handle toward feet
 - (7) Spread ribs: caution—lacerations from fractured ribs
 - (8) Evacuate clotted blood with towels/suction
 - (9) If injury on right side of chest, extend incision with shears or Gigli saw
 - c. Cardiac massage
 - (1) One handed
 - (a) Thumb over left ventricle
 - (b) Fingers over right ventricle
 - (c) Palm over apex
 - (d) Squeeze gently to compress.
 - (2) One hand and sternum
 - (a) Fingers together to form flat surface over left ventricle.
 - (b) Compress heart against sternum.

- (3) Two-handed: compressions superior to those in one-handed techniques
 - (a) Cup left hand, place over right ventricle.
 - (b) Use fingers of right hand to form flat surface and support left ventricle.
 - (c) Push flat surface of right hand to compress against cupped left hand.
- (4) Rate: most recommend 50–60 but no good evidence for this
- d. Pericardiotomy
 - (1) Perform if tamponade not excluded by ultrasound before procedure.
 - (2) Incise pericardium anterior and parallel to phrenic nerve.
 - (a) Lift with forceps, incise with scissors.
 - (b) Incise from apex to aortic root.
 - (c) Clear clots with sweeping motion of gloved hand or gauze.
- e. Control of cardiac wound hemorrhage
 - (1) If heart is beating, hold one finger over wound and use other hand to stabilize heart.
 - (2) If heart is not beating, perform repair, then resuscitate/defibrillate; perform intermittent cardiac massage during repair.
 - (a) Repair with staples (success rate 93% in one study) *or*
 - (b) Horizontal mattress sutures with 3-0 silk
 - (3) Place all sutures before tying to reduce tearing of myocardium
 - (a) Alternatives: use pledgets, *or* insert Foley catheter, inflate, place purse-string suture, deflate, remove, and tie suture
 - (b) Atrial wounds amenable to staples, Foley catheter technique
 - (c) Avoid ligation of coronary artery when possible.
- f. Great vessels wounds
 - (1) Control with digital pressure or partial-occlusion clamps
 - (2) Close small aortic wounds with 3-0 silk
 - (3) Left subclavian artery can be cross-clamped
 - (4) Right subclavian: use laparotomy pads to compress artery from apex of the pleural cavity from below and supraclavicular fossa from above while moving to the operating room
- g. Aorta cross clamping
 - (1) Indication: persistent hypotension (>70 mmHg) after thoracotomy and pericardiotomy
 - (2) Maintains myocardial/cerebral perfusion
 - (3) Beneficial with massive hemoperitoneum just before laparotomy
 - (4) Technique
 - (a) Identify aorta: lying on vertebral bodies
 - (b) Esophagus is medial and anterior to aorta (nasogastric tube helpful to identify).
 - (c) Expose aorta by elevating left lung, open pleura, and bluntly dissect esophagus from aorta, and aorta from pleura.
 - (d) Hold aorta in left index finger, and place clamp with right hand.
 - (5) Postresuscitation: systolic blood pressure 30 minutes after EDT predictive of outcome according to one study

- (a) Average systolic blood pressure of 110 mmHg in those with good neurologic recovery
- (b) Average systolic blood pressure of 85 mmHg in those survivors with significant neurologic deficits
- (c) Systolic blood pressure <70 mmHg: no survivors

III. PAIN MANAGEMENT

A. Acute pain management in adults

1. Pain in the emergency department
 - a. >60% of emergency department patients list pain as primary symptom.
 - b. Oligoanesthesia common problem in emergency department, especially noted for:
 - (1) Ethnic minorities
 - (2) Extremes of age
 - (3) Cognitively impaired
 - c. Barriers to pain control
 - (1) Patient: fear of addiction, acceptance of pain, unwillingness to ask
 - (2) Provider: prevalence of drug seeking patients, lack of education, inability to perceive alternative presentations of pain, lack of objective measurement tool
 - (3) System: lack of guidelines and standards
2. Opioid analgesia
 - a. Binds and stimulates receptors in brain and spinal cord
 - (1) $\mu 1$: supraspinal analgesia, euphoria, miosis, urinary retention
 - (2) $\mu 2$: respiratory and cardiovascular depression, decreased GI motility
 - (3) $\mu 2$ and cannabinoid receptors: thought to be responsible for addiction
 - (4) κ : dysphoria and spinal level analgesia
 - b. No pure $\mu 1$ agonists exist at this time.
3. Evaluation of pain
 - a. Required at triage by the Joint Commission
 - b. Includes assessment of location, severity, duration, quality, exacerbating and relieving factors
 - c. Pain scales
 - (1) Adjective rating scale: ordered list of descriptions, easy to administer
 - (2) Visual analog scale: scale with millimeter markings from no pain to worst pain imaginable
 - (3) Numeric rating with descriptors: useful for patients with visual or dexterity difficulties
 - (4) Numeric rating without descriptors: most commonly used, easy to administer
 - (5) Special populations
 - (a) Use family members to help evaluate signs of distress
 - (b) Visual analog scale most reliable with language and cultural differences

4. Treatment of pain

- a. Key: select agent appropriate for severity of pain, rapid onset, ease of administration, safety, and efficacy.
- b. Local infiltration: no systemic effects, limited duration
- c. Peripheral nerve block: technically difficult, but no systemic opioids, ultrasound useful adjunct
- d. Regional anesthesia: no opioid or general anesthesia but risk of systemic complications
- e. Classes
 - (1) NSAIDs: mild to moderate pain, colicky pain, oral, IM, IV, caution in elderly, decreased renal function
 - (a) Bind to cyclooxygenase 2 receptors
 - (b) Reduce inflammation
 - (c) Risk of sudden cardiac death with all NSAIDs but highest with COX-2 specific inhibitors
 - (d) Adverse effects: GI bleeding and upset, nephropathy, platelet dysfunction, dizziness, headaches
 - (e) May cause renal failure in elderly, volume depleted, cardiac disease, loop diuretics

Table 52: Opioids

Agent	Equipotent dose (mg) IV/IM/oral	Onset (min)	Duration (hours)	Comments
Morphine	10/10/60	1–2 IV	1–2 IV	Histamine release: transient nausea, emesis, hypotension
Hydromorphone	1.5/1.5/7.5	3–5 IV	2–4 IV	
Fentanyl	0.1/0.1/0.2 (transmucosal)	<1 IV	½–1 IV	High dose: chest wall rigidity
Oxycodone	NA/NA/30	10–15 oral	3–6 oral	Acetaminophen overdose with high doses
Hydrocodone	NA/NA/30	30–60 oral	3–5 oral	Acetaminophen overdose with high doses
Codeine	NA/NA/200	30–60 oral	Varies	Many GI effects

NA = not applicable

- (2) Opioids: moderate to severe pain, oral (slower onset but can be effective), IM (unreliable onset, painful), IV (painful once, easily titrated)
- (3) Opioid agonists–antagonists
 - (a) Reduce adverse effects (respiratory depression)
 - (b) Use with extreme caution in opioid addicted (may cause severe withdrawal)
- (4) Acetaminophen: effective for mild to moderate pain
 - (a) No effects on platelets
 - (b) No anti-inflammatory effects
 - (c) Maximum adult dosage 3 g/day
 - (d) No adjustment in renal or mild hepatic impairment

5. Neuropathic pain
 - a. Tricyclic and neuroleptic medications
 - (1) Amitriptyline: chronic pain
 - (2) Carbamazepine: trigeminal neuralgia
 - (3) Gabapentin and pregabalin: neuropathic pain
 - b. Ensure follow-up for titration
 - c. Dosing guidelines for opioids
 - (1) Titrate dosage to desired effect while minimizing adverse effects.
 - (2) Decrease initial dosage in elderly, respiratory impairment including obstructive sleep apnea
 - d. Renal and hepatic impairment: fentanyl, hydromorphone medications of choice
- B. Pain management in children and neonates
 1. Developmental stages affect approach to painful procedures
 - a. Neonate/infant (0–9 months old)
 - (1) No concept of time, so no anxiety
 - (2) Pain may affect future neurodevelopment
 - (3) No stranger anxiety
 - (4) Unable to abstract: brief painful procedure does not affect response to next procedure
 - b. Toddler (10 months to 3 years old)
 - (1) Stranger anxiety: place in lap of parent
 - (2) Want to make choices but lack logical thinking
 - c. School aged (4–10 years old)
 - (1) Independent thinking, can be engaged
 - (2) Short duration pain can be mitigated through counting or other activity.
 - (3) Can conceptualize time: understand expected length of pain
 - (4) Magical thinking: can work for or against provider
 - (5) Physical restraint not recommended
 - d. Preadolescence and adolescence (11–18 years old)
 - (1) Physical modesty important
 - (2) May regress when faced with painful procedure
 - e. Developmentally delayed
 - (1) May be a larger child who exhibits behavior of younger child
 - (2) Often have complex medical conditions requiring procedures
 - (3) Physical restraint may be dangerous to patient and provider.
 2. Painless emergency department toolbox
 - a. Choices: anxiolysis, analgesia, sedation
 - b. Considerations
 - (1) Patient: developmental level, anxiety level, airway issues
 - (2) Procedure: degree of pain, duration, need for motionless
 - (3) Provider: skill of physician, nurses, resources of emergency department

c. Anxiolysis

(1) Nonpharmacologic factors

- (a) Parental presence
- (b) Distraction
 - i. Young children: books, bubbles, music, light wands
 - ii. Older children: videos, guided imagery, video games
- (2) Pharmacologic agents: benzodiazepines, particularly midazolam
 - (a) Dosage: 0.5 mg/kg orally, 0.05 mg/kg IV
 - (b) Onset: 20 minutes oral, 2–3 minutes IV
 - (c) IV preparation can be given orally or intranasally.
 - (d) Pair with lidocaine/epinephrine/tetracaine for wound repair.

d. Analgesia

(1) Topical

- (a) EMLA (mixture of local anesthetics) or topical lidocaine anesthetic cream on intact skin for IV placement; wait 30 minutes
- (b) Lidocaine/epinephrine/tetracaine
 - i. Effective for wound into subcutaneous tissue
 - ii. Place ½ in wound, saturate cotton ball with remainder, and apply to wound with light pressure.
 - iii. Wait 20 minutes.
 - iv. Blanching of skin indicates effectiveness.

(2) Local: usually lidocaine

- (a) Slow injection with small (30-gauge needle)
- (b) Buffer with bicarbonate (1 mL bicarbonate to 4 mL lidocaine)
 - i. Regional blocks may be preferable in some cases
 - ii. Less tissue distortion, greater analgesia
- (c) Dosing limit: 3 mg/kg lidocaine, 5 mg/kg lidocaine with epinephrine

(3) Systemic

- (a) Sucralose 25% offered on pacifier for infants
- (b) Ketorolac IM or IV
 - i. Dosage 1 mg/kg IM, 0.5 mg/kg IV
 - ii. Approved for patients ≥ 2 years old
 - iii. Mild to moderate pain, especially musculoskeletal, renal stones, and ovarian pathology
- (c) Opioids for moderate to severe pain
 - i. Painful conditions should be treated based on condition not on patient appearance.
 - ii. Children require more opioids proportional to weight than adults.

Table 53: Specific Opioid Agents

Agent	Initial IV Dosage	Comments
Morphine	0.1–0.3 mg/kg	Histamine release: pruritus, nausea, hypotension
Hydromorphone	0.015–0.020 mg/kg	Pruritus and nausea
Fentanyl	1–2 mcg/kg	Bradycardia, chest wall rigidity in large doses, respiratory depression: may last longer than analgesia

e. Sedation (see also below)

(1) Indications

- (a) Very painful procedures of any length (eg, fracture reduction)
- (b) Moderately painful protracted (eg, incision and drainage of abscess)
- (c) Extreme anxiety/developmental delay when anxiolysis fails
- (d) Need for motionlessness

(2) Procedures not requiring routine sedation

- (a) Short, nonpainful procedures (eg, CT)
- (b) Very short, painful procedures (nursemaid's reduction)
- (c) Painful procedure when adequate analgesia can otherwise be provided

C. Procedural sedation and analgesia

1. Definitions

- a. **Procedural sedation:** administration of sedative or dissociative anesthetics to induce a depressed level of consciousness while maintaining cardiorespiratory function so that a medical procedure can be performed with little or no patient reaction or memory.
- b. **Analgesia** is the addition of agents to minimize pain.

2. Principles

- a. Determine level of sedation required.
- b. Ensure availability of appropriate monitoring and rescue equipment.
- c. Administer analgesics before sedatives.
- d. Titrate agents to level of sedation desired.
- e. Observe/monitor after procedure until at baseline.

3. Patient evaluation

- a. **Complication rate** determined by patient's current state of health and depth of sedation.
- b. **Focused history and physical examination** to identify potential issues (eg, COPD, intoxication, morbid obesity, hypovolemia)
- c. ASA classification useful in determining appropriateness for emergency department sedation (eg, Class III [patient with severe systemic disease] or worse may be inappropriate candidates for emergency department sedation).
- d. **Evaluate airway** for potential difficulty with bag-valve-mask or intubation (see pages 1116–1122); consider operating room sedation for patients with predicted difficult airway depending on clinical urgency.
- e. **Procedural urgency** may affect decision to delay sedation.

- (1) Emergent indications: cardioversion in unstable patients, neuroimaging in trauma, fracture reduction to restore circulation, care of contaminated wound
 - (2) Urgent: stable fracture reduction, incision and drainage, care of clean wound, foreign body removal, sexual assault examination
 - (3) Nonurgent: soft tissue foreign body removal, splinting
- f. Fasting
- (1) Risk of aspiration increases with depth of sedation; balance risk with urgency of procedure.
 - (2) Recommendations
 - (a) No oral intake >3 hours: any level of sedation
 - (b) Clear liquids in last 3 hours: risk slightly higher but still acceptable for any level of sedation
 - (c) Snack in last 3 hours: limit to moderate sedation if possible
 - (d) Meal in last 3 hours: limit to minimal sedation for nonurgent sedation
 - (3) Pediatrics: no correlation between fasting and sedation
 - (a) Study of 30,000 sedations: one aspiration in patient who had fasted 8 hours
 - (b) Vomiting common in recovery phase
4. Sedation levels
- a. Minimal: anxiolysis, no effect on airway, ventilation, cardiovascular function
 - b. Moderate: purposeful response to verbal/tactile stimulation, none to minimal effects on airway, breathing, cardiovascular system; dissociative sedation (ketamine) classified as moderate.
 - c. Deep: purposeful response only to deep pain; may affect airway, respirations
 - d. General: no response, often requires airway, ventilatory support, may need cardiovascular support (ie, for hypotension)
 - e. Most emergency department sedations are moderate; deep may be required for some procedures.
5. Step-by-step
- a. Preparation
 - (1) Equipment (airway adjunct, bag-valve-mask, suction, etc, medications, IV fluids)
 - (2) Personnel
 - (a) Anesthesia model calls for two physicians (one for sedation, one for procedure).
 - (b) Existing evidence shows emergency physicians can perform both for moderate sedation.
 - (c) Nurse or respiratory therapist can monitor patient while procedure performed by physician.
 - b. Monitoring: for moderate sedation
 - (1) Blood pressure every 5 min and after medication bolus
 - (2) Heart rate: continuous
 - (3) Respiratory rate: continuous
 - (4) O₂ saturation: continuous
 - (5) Capnography: consider continuous

- c. Preprocedural pain management
 - (1) Administration of opioids before procedure reduces pain during procedure
 - (2) Short-acting agents (fentanyl) preferred
- d. Oxygen administration
 - (1) Safe
 - (2) Not shown to effect incidence of desaturation
 - (3) May delay recognition of hypoventilation
- 6. Agents
 - a. Minimal sedation: nitrous oxide, midazolam, fentanyl, pentobarbital
 - b. Brief moderate/deep: methohexital, propofol, etomidate, ketamine
 - c. Extended: propofol, ketamine

Table 54: Common Agents

Agent	Initial Dosage (IV)	Onset (min)	Duration (min)	Comments
Midazolam	0.05–0.1 mg/kg	1–3	60	Can be combined with other agents, hypotension, long duration, paradoxical effect in some
Etomidate	0.15 mg/kg	1	5–10	Minimal cardiovascular effects, may suppress adrenal cortex, consider alternative agents in seriously ill patients, no analgesic effects, may cause myoclonic jerks
Propofol	1 mg/kg	1–2	5–10	Easy to titrate, respiratory depression, hypotension in hypovolemia, contains egg and soy
Ketamine	1 mg/kg	1–3	10–20	Dissociative amnesia and analgesia, no respiratory depression, emergence reaction, laryngospasm, hypersalivation, avoid in increased IOP, mixed evidence in increased ICP

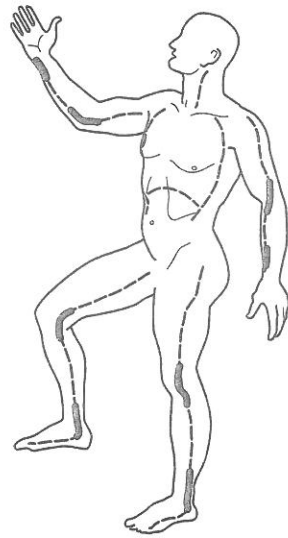
- 7. Elderly patients
 - a. Increased risk of adverse events
 - (1) Reduced cardiovascular reserve
 - (2) Underlying chronic respiratory/cardiovascular illnesses
 - (3) Anatomical challenges to airway intervention
 - b. Etomidate generally good choice, but avoid in patients with clonus at baseline.
 - c. Propofol: use ½ of recommended adult dose
 - d. Benzodiazepines/barbiturates: increased neurologic effects, especially when used with opioids

8. Complications
 - a. 2.3%–7.7% of procedural sedation/analgesia cases
 - b. Serious: airway interventions, hypotension, cardiac arrhythmias
 - c. Minor: transient hypoxia, emesis, deeper than desired sedation
9. Discharge
 - a. Observe after procedure until at baseline
 - b. Occurrence of adverse effects >5 minutes after procedure <1%
 - c. No reports of adverse events after discharge

IV. CUTANEOUS DIAGNOSTIC AND THERAPEUTIC PROCEDURES

A. Escharotomy

1. Indications: circumferential burns of the neck, chest, extremities that compromise airway access, ventilation or circulation
2. Technique: incise through the skin into the subcutaneous tissue
 - a. Sterile prep
 - b. Local or systemic anesthesia not required for full-thickness burns but may be necessary for partial-thickness burns
 - c. Bleeding may be controlled with direct pressure or electrocautery
 - d. Pack wounds with sterile gauze with antibiotic ointment
3. Locations
 - a. Neck
 - (1) Incise the lateral aspect of the neck from clavicle to mastoid process
 - (2) Posterior to the carotid arteries
 - b. Chest wall
 - (1) Incise along the anterior axillary line from the second to the twelfth rib on each side.
 - (2) Connect the two incisions transversely.
 - c. Extremities
 - (1) Incise on the medial and lateral aspect of the limb
 - (2) 1 cm proximal to the burn to 1 cm distal to the burn
 - (3) Incisions may be extended to the thumb and fingers/toes
 - (4) Incisions should cross joints: take care to avoid ulnar, radial, superficial peroneal, and posterior tibial nerves.
 - (5) Improved color, Doppler flow, sensation, pulse oximetry indicate restored flow.



Escharotomy Sites

4. Complications of procedure
 - a. Bleeding
 - b. Infection
 - c. Damage to underlying structures
 5. Complications of inadequate decompression
 - a. Local: muscle necrosis, nerve injury, amputation
 - b. Systemic: myoglobinuria/renal failure, hyperkalemia, metabolic acidosis
- B. Incision and drainage**
1. Abscess formation
 - a. Local superficial cellulitis infection produces necrosis/liquefaction
 - b. Walling off of leukocytes and debris = abscess
 - c. MRSA 60%–75% of all abscesses
 - d. Oral, rectal, vulvovaginal abscesses more likely polymicrobial
 2. Diagnosis
 - a. Usually visual
 - b. Needle aspiration
 - c. Ultrasound highly accurate



caption for this image??

- d. Plain radiographs not recommended except if foreign body suspected (IV drug use needles)
- 3. Setting for incision and drainage
 - a. Emergency department: most abscesses
 - b. Operating room
 - (1) Large or deep space abscesses
 - (2) Abscesses near neurovascular structures
 - (3) Inability to obtain adequate anesthesia
 - (4) Most hand abscesses except distal fingers
- 4. Preparation
 - a. Position patient, prep and drape
 - b. Universal precautions, especially face shield
 - c. Equipment: scalpel, hemostats, packing, dressing
 - d. Anesthesia
 - (1) Local anesthesia usually adequate
 - (2) Lidocaine
 - (a) Approach abscess from the side
 - (b) Infiltrate skin over abscess
 - (c) Infiltrate deeper until wall of cavity felt
 - (d) Distend cavity with several milliliters of lidocaine (lessens pain of packing)
- 5. Procedure
 - a. #11 or #15 scalpel
 - b. Incise along skin lines
 - c. Extend incision to edges of abscess
 - d. Express pus with gentle compression
 - e. Deloculate with hemostats covered in gauze or a cotton swab
 - f. No evidence that irrigation is useful
 - g. Pack with plain gauze
 - (1) Not shown to be useful for small abscesses
 - (2) Packing should be removed in 2–3 days, replaced if active drainage
 - h. Dress with absorbent gauze
 - i. Obtain cultures for systemically ill patients
- 6. Antibiotics
 - a. Not indicated in simple cutaneous abscesses
 - b. Indications for oral antibiotics – coverage for MRSA
 - (1) Multiple abscesses
 - (2) Significant surrounding cellulitis
 - (3) Immunocompromised patients
 - c. Parenteral antibiotics
 - (1) Systemic illness
 - (2) Extensive cellulitis

- d. Endocarditis prophylaxis (before procedure) indicated for high-risk patients
 - (1) Prosthetic valve/repair material
 - (2) Unrepaired CHD/ or repaired with residual defects
 - (3) Cardiac transplants with valvulopathy
- 7. Complications of incision and drainage
 - a. Residual numbness
 - b. Injury to neurovascular structures
 - c. Scarring
 - d. Delayed or poor wound healing in diabetes mellitus or peripheral vascular disease
- C. Trephination, nails
 - 1. Indication: subungual hematoma >50% of nail bed surface
 - 2. Good to excellent outcome with trephination regardless of hematoma size, mechanism of injury, or presence of distal tuft fracture
 - 3. Methods
 - a. Handheld electrocautery: preferred method
 - (1) Rapid and painless
 - (2) Do not apply alcohol to nail (flames may result)
 - b. Heated paper clip
 - (1) May cause coagulation of hematoma
 - (2) Introduces carbon particles into nailbed
 - (3) Delayed healing, tattooing
 - c. 18-gauge needle or scalpel
 - (1) Painful
 - (2) May require digital block
 - 4. Care after procedure: soak wound in warm, soapy water 2–3 times daily × 7 days
- D. Wound closure techniques
 - 1. Wound tape
 - a. Advantages: least tissue reactive, lowest infection rates, rapid, painless, low cost, no return visit for removal
 - b. Disadvantages: fall off, low tensile strength, highest rate of dehiscence, cannot get wet, cannot be used in hair, toxic adjunct required for adherence
 - c. Use limited to:
 - (1) Low tension simple wounds
 - (2) Skin tears
 - d. Technique
 - (1) Apply tincture of benzoin to either side of wound edge, dry until tacky.
 - (2) Apply tape perpendicular to wound edge.
 - (a) Start in center, apply strips away from center on either side.
 - (b) Leave 2–3 mm space between.
 - (c) Apply additional pieces of tape across ends of strips overlying wound.
 - 2. Cyanoacrylate tissue adhesives
 - a. Advantages: rapid, painless, resist bacteria, occlusive, no need to remove, low cost

- b. Disadvantages: lower tensile strength than 5.0 or larger suture, dehiscence over high tension areas, cannot use on hands, can shower but not bathe or swim
 - c. Use limited to
 - (1) Relatively low tension wounds
 - (2) Wounds with edges easily approximated with fingers, forceps
 - (3) Hemostatic wounds
 - (4) Do not use on hands or over joints
 - d. Technique
 - (1) Approximate wound edges with nondominant hand (or by assistant)
 - (2) Use wound tape at intervals to approximate edges of large wounds
 - (3) Glue over wound and tape
 - (4) Octyl-cyanoacrylate
 - (a) Brush over wound surface parallel to wound in continuous motion.
 - (b) Allow to dry 30–45 seconds.
 - (c) Apply second layer.
 - (5) Butyl-cyanoacrylate
 - (a) Deposit glue in discreet drops or along entire wound.
 - (b) Single layer only
3. Staples
- a. Advantages: rapid, low tissue reactivity, low cost, removal less painful than sutures
 - b. Disadvantages: requires anesthesia, requires removal, less meticulous closure than sutures, may interfere with imaging
 - c. Limit use to:
 - (1) Linear, non-facial lacerations
 - (2) Excellent for scalp lacerations not through galea
 - d. Technique
 - (1) Approximate and evert wound edges with fingers or forceps.
 - (2) Align centerline indicator of stapler with center of wound.
 - (3) Touch stapler lightly to skin, and deposit staple; firm pressure may cause depression of wound edges.
 - (4) Pull stapler slightly backward to disengage.
 - (5) Place staples 2–4 mm apart.
4. Sutures
- a. Advantages: greatest tensile strength, meticulous closure, lowest dehiscence rate
 - b. Disadvantages: slow, requires anesthesia, greatest tissue reactivity, highest cost
 - c. Sizes
 - (1) Larger: stronger, more tissue damage and greater scar
 - (2) Smaller: less strong, less scarring
 - d. Types
 - (1) Nonabsorbable (eg, nylon, polypropylene, polybaster, silk)
 - (a) Maintain tensile strength for at least 60 days
 - (b) Best for skin closure, tendon repair

- (c) Avoid in deep tissues (foreign body reaction)
- (d) Polybaster sutures: can elongate, good for wounds with anticipated swelling
- (2) Absorbable (eg, gut, chromic gut, coated vicryl)
 - (a) Best suited for closure of dermis and fascia
 - (b) Fast absorbing (eg, fast absorbing gut, Vicryl Rapide), useful for superficial skin closure when suture removal not desirable
- e. Techniques
 - (1) Simple, interrupted percutaneous
 - (a) Needle introduced to skin, exit through dermis into wound. then reinsert at level of dermis on opposite side of wound, exit through skin.
 - (b) Enter and exit skin at equal distance from wound: deeper "bite" improves wound eversion
 - (c) Tie with square knots: number of knots = suture size
 - (d) Pull knots to one side of wound.
 - (e) Place first suture in center, next sutures on either side, bisecting distance from first suture to wound edge, etc.
 - (2) Continuous (running) percutaneous
 - (a) Appropriate for long linear lacerations
 - (b) Initial suture similar to interrupted
 - (c) Do not tie, reinsert needle on opposite side: cross wound with suture at 65° angle
 - (d) Bring needle out on opposite side, suture under skin perpendicular to wound.
 - (e) Repeat and tie.
 - (3) Deep dermal
 - (a) Reduces tension on wound
 - (b) Closes dead spaces
 - (c) Not useful in adipose tissue
 - (d) Insert needle at mid dermis, exit below epidermal/dermal junction.
 - (e) Reinsert needle on other side of wound at epidermal/dermal junction, exit at mid dermis, and tie (knot is buried).
 - (4) Continuous subcuticular
 - (a) Complex; rarely used in emergency department
 - (b) Usually absorbable suture material (no removal)
 - (c) Anchor knot in deep dermis.
 - (d) Take sequential horizontal bites on each side, just below epidermal/dermal interface until wound closed.
 - (5) Horizontal half-buried mattress (corner stitch)
 - (a) Particularly useful for closing tip of skin flaps because do not cut off blood supply
 - (b) Insert needle through skin at one side of wound.
 - (c) Insert horizontally near tip of flap at level of dermis.
 - (d) Exit through dermis of flap at opposite edge (do not break through skin of flap).
 - (e) Insert needle into dermis on opposite side of wound.
 - (f) Exit through skin and tie knot.

- (6) Vertical mattress
 - (a) Results in excellent wound edge eversion
 - (b) Useful in deep wounds when subcutaneous tissue too friable for deep sutures
 - (c) Useful in lax or thin skin
 - (d) Take a large, deep bite through each side of wound; entering the skin from the same side of the wound just exited, take a superficial bite back through the wound, and tie knot.
 - (e) Alternative method that places less tension on superficial stitch: make a superficial closure near to the wound edges, pull the suture away from the wound (elevates wound edges), throw deep suture farther away from wound edge, and tie knot.

E. Wound management

1. Skin and fascia anatomy

- a. Epidermis: several layers, most important
 - (1) Stratum germinativum: new skin cells produced
 - (2) Stratum corneum: outermost, gives skin cosmetic appearance
- b. Dermis: thickest layer, connective tissue
 - (1) Key layer for ultimate healing of wounds
 - (2) Must remove debris/devitalized tissue
- c. Superficial fascia
 - (1) Below dermis, encloses subcutaneous fat
 - (2) Irrigate and debride to reduce infection rate
- d. Deep fascia
 - (1) Beneath fat, protects muscle
 - (2) Must be closed to prevent spread of infection

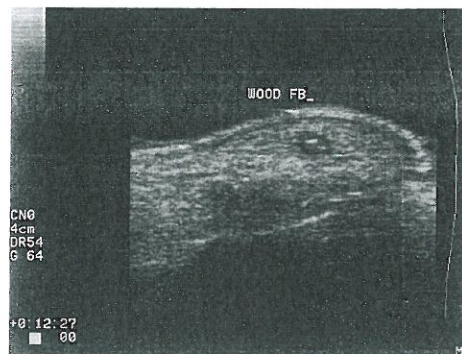
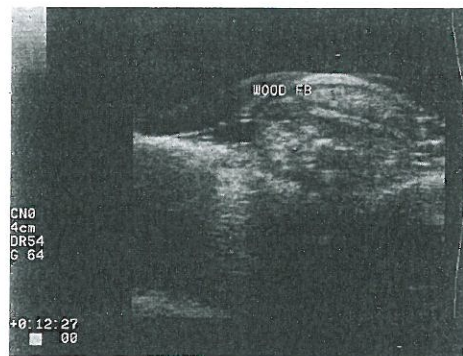
2. Wound morbidity risk factors

- a. Underlying medical conditions: peripheral vascular disease, diabetes mellitus, immunocompromised states.
- b. Mechanism and setting
 - (1) Contaminated wounds
 - (2) Foreign body involvement
 - (3) Crush-type injuries
- c. Tetanus status

3. Risk factors for infection

- a. Injury >8–12 hours old
- b. Location: leg/thigh > arms > feet > chest > back > face > scalp
- c. Contamination: foreign body, saliva, feces
- d. Crush mechanism
- e. Presence of subcutaneous sutures
- f. Type of repair: suture > staples > tape
- g. Anesthesia with epinephrine
- h. High-velocity projectile injuries

4. Primary versus secondary closure
 - a. Decision based on:
 - (1) Time from injury
 - (2) Location of injury (face and scalp up to 24 hours, other areas 8–12 hours)
 - (3) Degree of contamination
 - (4) Underlying conditions affecting circulation
 - (5) Cosmesis
 - b. Wounds not closed primarily because of risk of infection should be reevaluated for delayed primary closure after 4 days.
5. Wound examination
 - a. Requires adequate analgesia and visualization
 - b. May be appropriate to extend incisions for evaluation
 - c. Wounds that cannot be explored fully or with probable foreign body
 - (1) Additional imaging
 - (2) Expert consult for immediate or delayed surgery in operating room
 - d. Foreign body examination
 - (1) Adult patients with foreign body sensation – more likely to have retained foreign body than those who do not report foreign body sensation (LR 2.49 and 0.69)
 - (2) No single method guarantees identification and removal of all foreign bodies.
 - e. Imaging studies
 - (1) Plain radiographs: useful only for radiopaque foreign bodies, glass >1–2 mm thick may be visualized, organic foreign bodies not seen
 - (2) CT: excellent but expensive and increased radiation
 - (3) Ultrasound: can visualize organic and metallic foreign bodies, operator dependent, may be difficult with distorted tissue planes



6. Wound preparation

a. Full sterile technique not required; clean nonsterile gloves are as effective as sterile gloves in infection prevention.

b. Anesthesia

(1) Provide before extensive examination and preparation

(2) Local/regional

(a) Lidocaine most common

i. Local: onset seconds, duration 20–60 minutes

ii. Regional: onset 4–6 min, duration 60–75 minutes

iii. Maximal dose 35 mg/kg

iv. Can add epinephrine

- Improved hemostasis
- Increases infection risk and delays healing
- Recent literature suggests safe in digital blocks in patients without underlying peripheral vascular disease, Raynaud disease
- Maximal dosage 5–7 mg/kg

(b) Bupivacaine

i. Onset slower than lidocaine

ii Duration 8 times that of lidocaine

iii Maximum dosage 2.5 mg/kg without epinephrine, 3.5 mg/kg with epinephrine

(3) Topical agents

(a) Lidocaine/epinephrine/tetracaine

(b) EMLA (mixture of local anesthetics)

c. Skin preparation

(1) Disinfect skin around wound but not wound itself: chlorhexidine (preferred) or povidone-iodine

(2) Hair removal

(a) Not necessary to reduce risk of infection

(b) May facilitate suture placement and removal

(c) Do not remove where hairline cosmesis important (eg, eyebrow)

(d) Shaving: 3–9 times greater risk of infection than clipping

d. Irrigation

(1) Decreases bacteria count, reduces risk of infection

(2) Need for routine irrigation questionable for simple, nonbite, noncontaminated wounds on scalp or face

(3) Low-pressure irrigation

(a) 0.5 psi

(b) Sufficient for uncontaminated wounds and loose tissue (eyelids, scrotum)

(4) High-pressure irrigation ~7 psi

(a) Wounds with high risk of infection

(b) Use 18-gauge angiocatheter with 50-mL syringe

(5) Irrigation with tap water as effective as with sterile solutions

- e. Debridement/ foreign body removal
 - (1) Debridement most important aspect of wound care
 - (a) Devitalized tissue delays healing, increases infection risk
 - (b) But – may result in wider scar
 - (c) Large areas of questionably viable skin or muscle: prep for delayed primary closure
 - (2) Foreign body removal
 - (a) Forceps
 - (b) Irrigation
 - (c) Probing wound with gloved hand not recommended
- 7. Prophylactic antibiotics
 - a. No clear evidence that prophylactic antibiotics routinely reduce wound infections
 - b. Recommended for:
 - (1) Crush injuries
 - (a) Start antibiotics before or immediately after wound manipulation.
 - (b) No benefit of continuing 24 hours beyond repair.
 - (c) Agent depends on likely pathogens.
 - (2) Contaminated or wounds with retained devitalized tissue
 - (3) Human bites to hands, feet, or those overlying joints
 - (4) Wounds contaminated by fresh water and plantar puncture wounds: cover for *Pseudomonas*
 - (5) Cat bites
 - (6) Dog bites (controversial)
 - (7) Intraoral lacerations
 - (8) Some authors advocate antibiotics for immunocompromised patients and those with prosthetic joints and valves, although evidence is lacking
- 8. Closure (see pages 1146–1149)
- 9. Dressings
 - a. Optimal: gas permeable, nonadherent, absorb some fluid but not allow dessication, outer layer impermeable to bacteria
 - b. Foams/gels/Vaseline-impregnated gauze all acceptable
 - (1) Foam/gel: can leave on 7 days
 - (2) Vaseline-impregnated gauze: 1–2 days
- 10. Immobilization: wounds in proximity to joints should be immobilized/splinted
 - a. Hastens healing
 - b. Decreases lymph flow (prevents spread of infection)
- 11. Tetanus immunization
 - a. Many people are under-immunized (elderly, immigrants, limited education).
 - b. Tetanus with diphtheria toxoid with acellular pertussis (Tdap) now recommended in adolescents and adults without previous booster.
 - c. Inadequately immunized patients should also get a dose of tetanus immunoglobulin with Tdap: provides antibodies for 4 weeks.

12. Aftercare

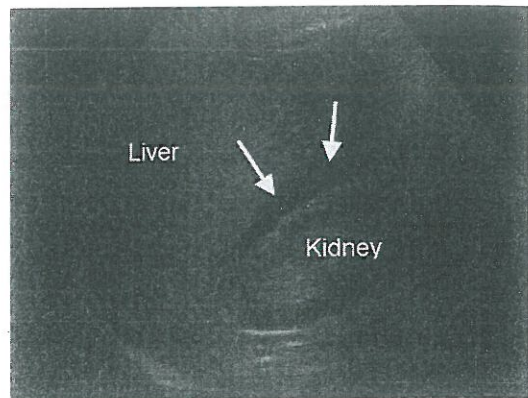
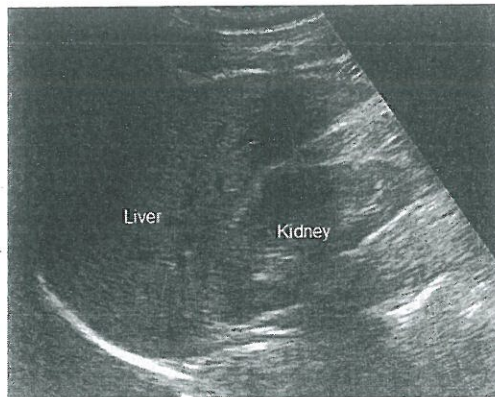
- a. Must provide instructions for wound care, signs of infection, follow-up (48 hours for high-risk wounds)
- b. Suture removal
 - (1) Face 3–5 days (replace w wound tape)
 - (2) Scalp, trunk 7–10 days
 - (3) Extremities 10–14 days
 - (4) Joints 14 days

V. ULTRASOUND**A. Bedside ultrasound**

- 1. Focused rather than comprehensive examination
- 2. Physician both performs and interprets
- 3. Answers clinical question or provides clinical guidance
- 4. Primary limitation is experience/skill of provider
- 5. Emergency department indications: trauma, abdominal aortic aneurysm (AAA), first trimester, cardiac, deep-vein thrombosis, gallbladder, renal, ultrasound-guided procedures
- 6. Physics
 - a. Ultrasound = sound waves >20,000 Hz; scanning usually between 2 and 20 MHz
 - b. Principles
 - (1) Sound waves projected into tissues from transducer
 - (2) Strike reflective surfaces and bounce back to transducer
 - (3) Image on screen made up of reflected waves
 - (a) Location: time the wave traveled before returning; deep structures in far field of screen
 - (b) Brightness: strength of returning wave; dense tissues have brighter reflection
 - c. Kinetic energy: use ALARA (as low as reasonably possible) energy to perform studies
 - d. Transducer frequency
 - (1) High
 - (a) Good resolution, poor penetration
 - (b) Use for peripheral/superficial structures (lines)
 - (2) Low
 - (a) Poor resolution, high penetration;
 - (b) Use for deep structures (abdomen, FAST)
 - e. Modes
 - (1) B-mode (brightness) : standard scanning
 - (2) M-mode (motion): shows one slice of image over time; fetal heart tones, cardiac motion
 - (3) Doppler: velocity of moving structures

B. Focused assessment with sonography in trauma (FAST)

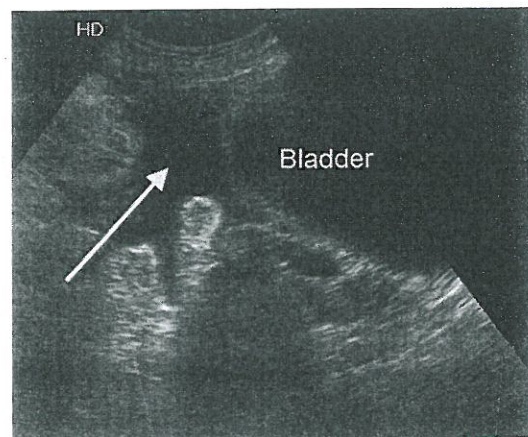
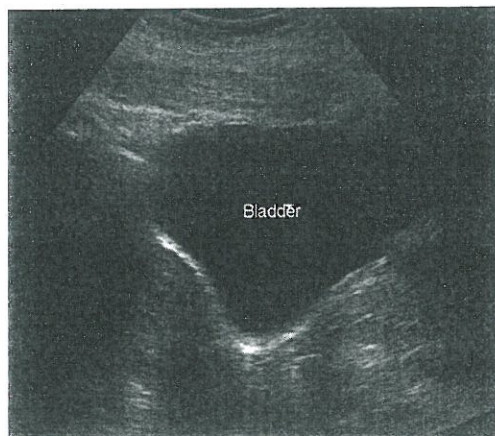
1. Goal: identify hemoperitoneum, hemopericardium
 - a. Sensitivity 60%–99% (range varies due to gold standard comparison differences)
 - b. Specificity 80%–99%
2. Extended FAST: includes identification of hemothorax, pneumothorax
3. Windows
 - a. Right upper quadrant (Morison's pouch), left upper quadrant, pelvis, cardiac



Courtesy of Sandra L. Werner, MD, RDMS, FACEP

Image on the left demonstrates a normal right upper quadrant view of the liver abutting the kidney. In the image on the right, the white arrows point to free fluid (blood) in Morison's pouch, the space between the liver and the kidney.

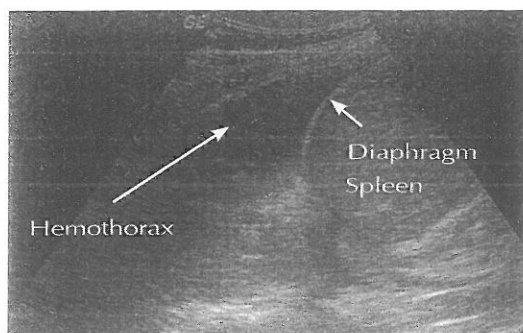
- b. Free fluid seen as black (anechoic)
 - (1) Requires ~250 mL for positive FAST
 - (2) Free fluid may be blood, urine, bile, ascites



Courtesy of Sandra L. Werner, MD, RDMS, FACEP

Image on the left shows a normal filled bladder in the sagittal plane. In the image on the right, the white arrow points to free fluid (blood) within the peritoneum.

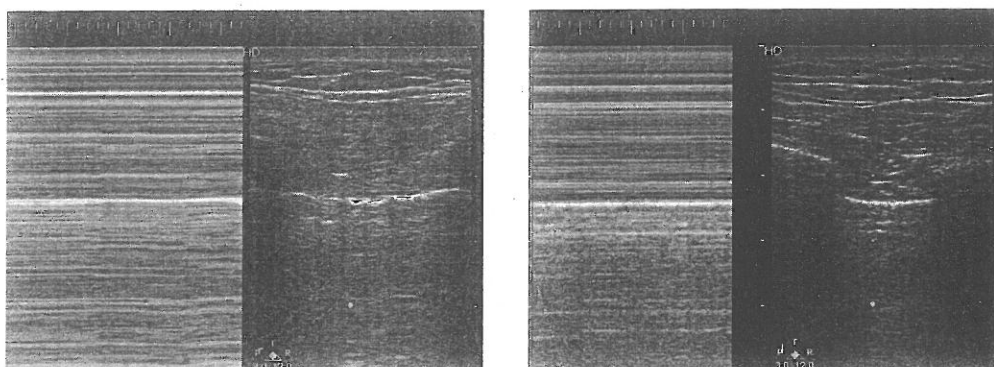
- c. Hemothorax
 - (1) Use right and left upper quadrant views and look superior to diaphragm
 - (2) Anechoic fluid above diaphragm = hemothorax



Courtesy of Sandra L. Werner, MD, RDMS, FACEP

d. Pneumothorax

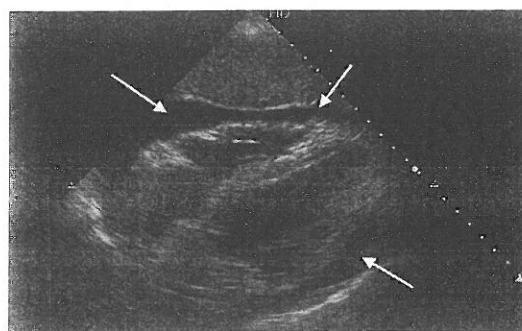
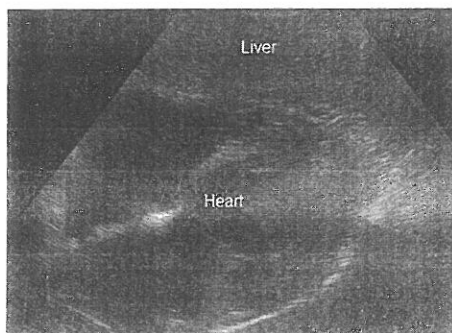
- (1) Use low- or high-frequency transducer.
- (2) Place on anterior chest wall in sagittal plane at second intercostal space.
- (3) Examine for movement of pleura.
- (4) Often demonstrated in M-mode
- (5) Ultrasound superior to supine chest radiograph for diagnosing pneumothorax.



Courtesy of Sandra L. Werner, MD, RDMS, FACEP

The image on the left demonstrates normal lung sliding in M-mode. A speckled pattern (seashore sign) is seen below the pleura (bright white line), consistent with normal lung sliding. In the image on the right, there are multiple straight lines beneath the pleura (stratosphere sign), indicating a lack of lung sliding and a pneumothorax.

- #### e. Cardiac: may use subxiphoid (transducer at xiphoid notch, beam directed superiorly through liver), or parasternal (transducer left of sternum at second or third intercostal space)



Courtesy of Sandra L. Werner, MD, RDMS, FACEP

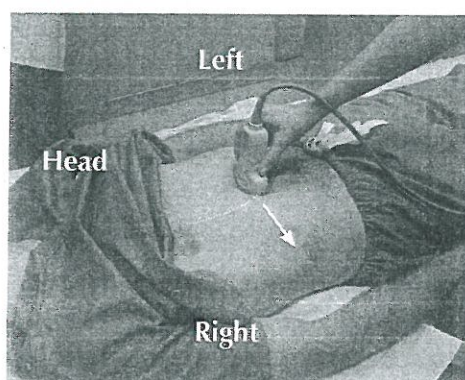
Image on the left shows a normal subxiphoid view with a single echogenic (white) line surrounding the heart. In the image on the right, an effusion is seen; the white arrows point to the anechoic (black) fluid surrounding the heart.

4. Clinical utility

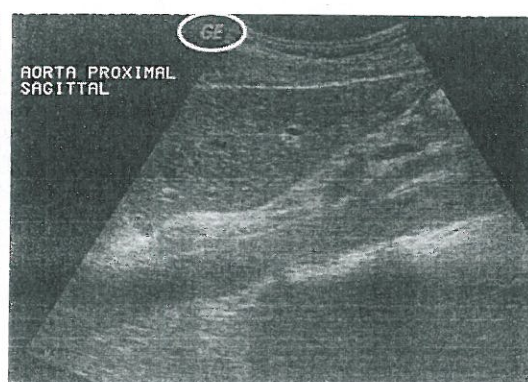
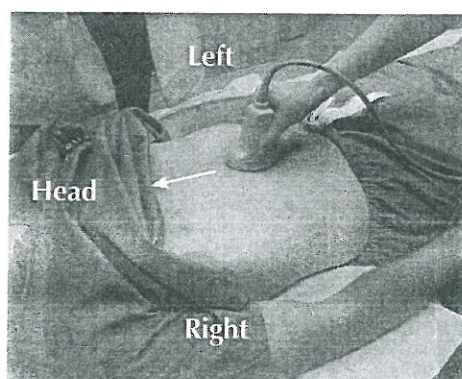
- a. Unstable patients with grossly positive FAST should go to the operating room.
- b. FAST does not identify all injuries (eg, bowel perforation, solid organ injuries without hemoperitoneum, retroperitoneal injuries).
- c. Ultrasound shown to reduce mortality and time to operating room in traumatic pericardial effusions.
- d. Accuracy of FAST in children similar to that in adults, but many pediatric solid organ injuries are managed nonoperatively.
- e. FAST also shown to be useful in diagnosis and management of unstable medical patients.

C. Aortic ultrasound

1. Goal: identify AAA
2. Indication: elderly patients with abdominal, flank, back pain
3. Technique
 - a. Low-frequency transducer
 - b. Patient supine, aorta is (patient's) left of spine
 - c. Scan from diaphragm to iliac artery bifurcation in sagittal and transverse planes.
 - (1) Most AAA are infrarenal.
 - (2) Saccular aneurysms can be missed if only sagittal plane used.

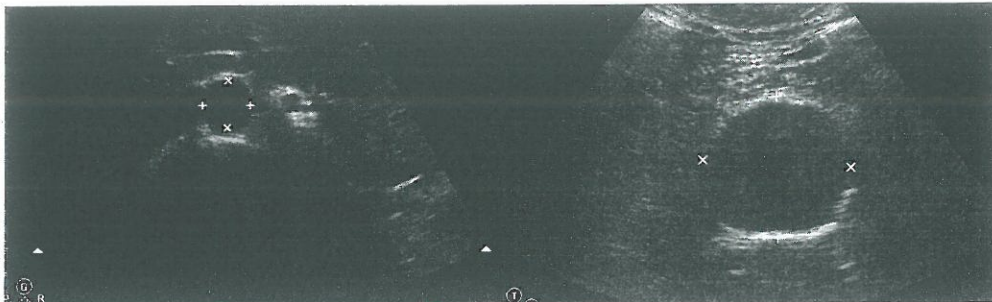


Courtesy of Sandra L. Werner, MD, RDMS, FACEP



Courtesy of Sandra L. Werner, MD, RDMS, FACEP

4. Diagnosis
 - a. AAA: aorta diameter >3 cm
 - b. Risk of rupture >5 cm
 - c. If AAA present, examine Morison's pouch for free fluid.
5. Clinical utility
 - a. Emergency physician-performed ultrasound highly accurate in diagnosing AAA.
 - b. Screening for AAA in the emergency department shown to be effective as well.

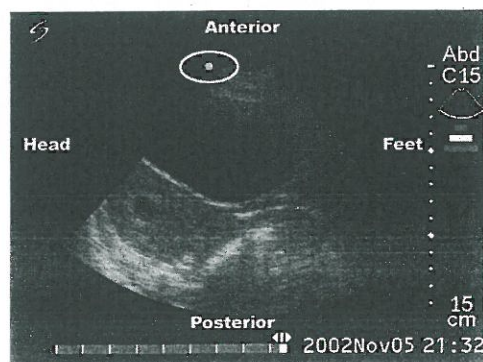
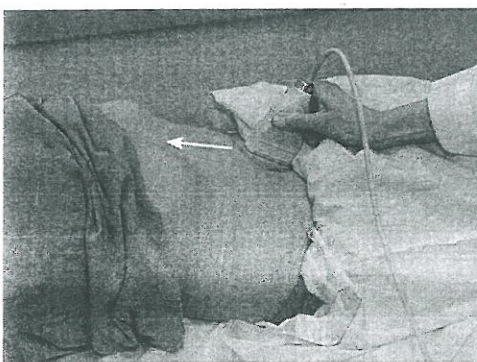


Courtesy of Sandra L. Werner, MD, RDMS, FACEP

The image on the left demonstrates the normal transverse view of the aorta. The image on the right demonstrates a large AAA.

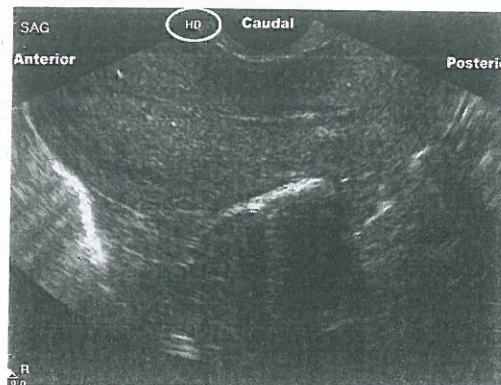
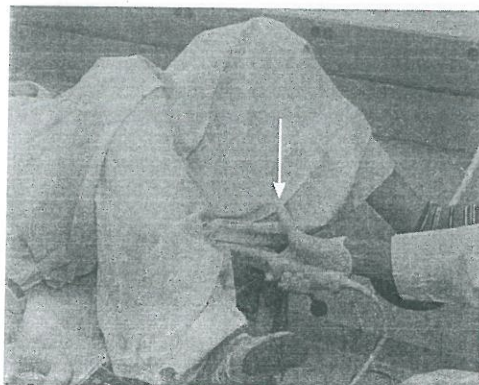
D. Pelvic ultrasound

1. Primary goals
 - a. Confirm intrauterine pregnancy
 - b. Identify ectopic pregnancy or findings suspicious for ectopic pregnancy
2. Secondary goals
 - a. Identify fetal viability or demise
 - b. Identify molar pregnancy
 - c. Identify ovarian pathology
3. Technique
 - a. Transabdominal (low-frequency abdominal transducer)
 - (1) Scan uterus in sagittal and transverse planes: full bladder to optimize view



Courtesy of Sandra L. Werner, MD, RDMS, FACEP

- (2) If free fluid noted, scan Morison's pouch
- b. Transvaginal (endocavitary transducer)
 - (1) Indicated if unable to visualize intrauterine pregnancy on transabdominal
 - (2) Scan uterus in sagittal and coronal views
 - (3) Empty bladder for optimal view
 - (4) Can detect 7 mL of free fluid in posterior cul-de-sac
 - (5) Has replaced culdocentesis for diagnosis of fluid in cul-de-sac

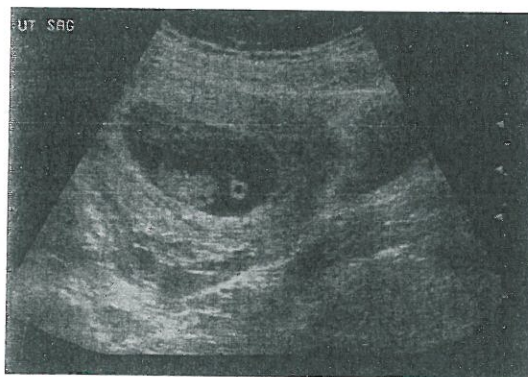


Courtesy of Sandra L. Werner, MD, RDMS, FACEP

4. Findings

a. Intrauterine pregnancy

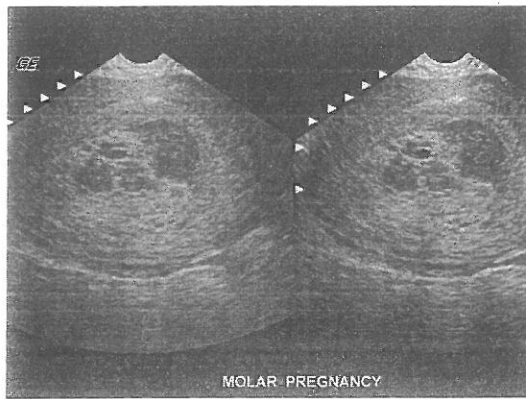
- (1) YS or fetus within fundus of uterus
- (2) Fetal demise: fetal pole >5 mm with no heart rate, or gestational sac >20 mm mean sac diameter without fetal pole



Courtesy of Sandra L. Werner, MD, RDMS, FACEP

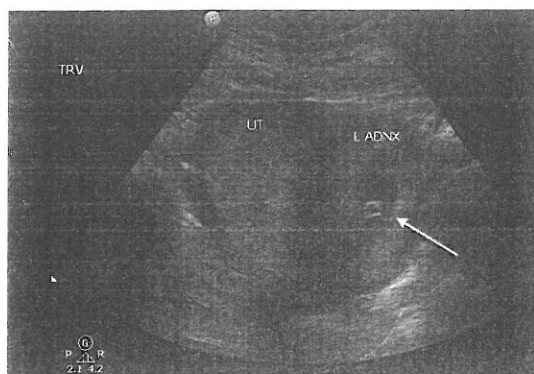
Normal transabdominal view of intrauterine pregnancy with fetus and yolk sac

b. Mole: echogenic, cystic appearance of endometrium



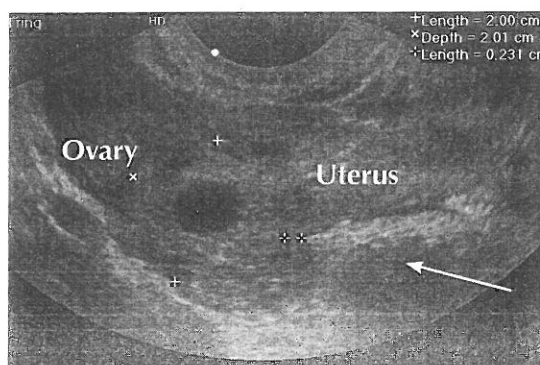
Courtesy of GE

c. Ectopic: chorionic ring or gestational sac with YS or fetus outside uterus or in cervix or cornu of uterus



Courtesy of Sandra L. Werner, MD, RDMS, FACEP

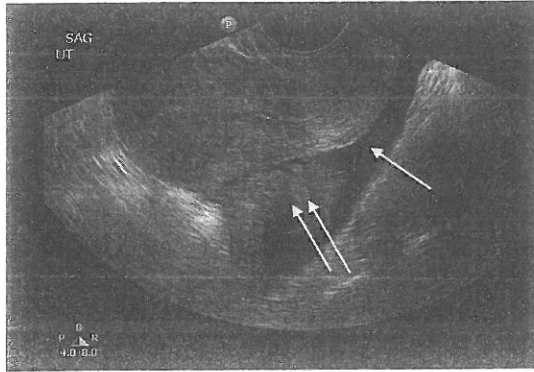
Transabdominal view of ectopic pregnancy with yolk sac (arrow)



Courtesy of Jessica Resnick, MD, RDMS, FACEP

Transvaginal view of tubal ring (arrow) between the ovary and uterus

d. Suggestive of ectopic: ovarian mass, simple or complex free fluid (clotted blood)



Courtesy of Sandra L. Werner, MD, RDMS, FACEP

Transvaginal view of free fluid (single arrow) and clotted blood (double arrow)

e. Indeterminate: no clear intrauterine pregnancy, no definite evidence of fetal demise/miscarriage, no evidence of ectopic

(1) If HCG high (>2,000) – obstetrics consult

(2) If HCG low – close follow-up with repeat ultrasound and HCG

5. Clinical utility: EP performed pelvic ultrasound demonstrated to:

- a. Reduce morbidity of ectopic pregnancies by decreasing time to diagnosis and OR
- b. Increase rate of early detection of ectopic pregnancies
- c. Reduce emergency department throughput times
- d. Increase patient satisfaction

E. Cardiac ultrasound

1. Indications: cardiac arrest, hypotension, suspicion for effusion/tamponade

2. Views

- a. Subxiphoid transverse is standard trauma view, sagittal useful for evaluating IVC, right atrium
- b. Parasternal long and short axis: transducer placed to the left of the sternum at the second or third intercostal space: evaluation of global EF, effusions
- c. Apical: transducer at PMI, angled superiorly toward left shoulder: evaluation of global EF, effusions, chamber sizes

3. Clinical utility

a. Cardiac arrest

(1) Detect effusion/tamponade

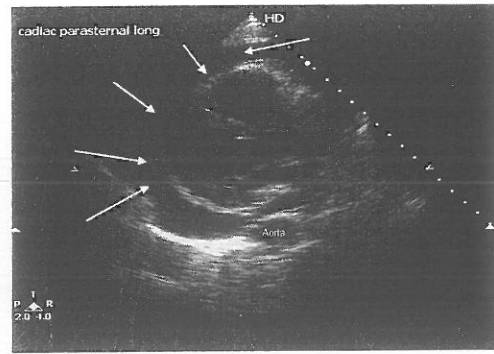
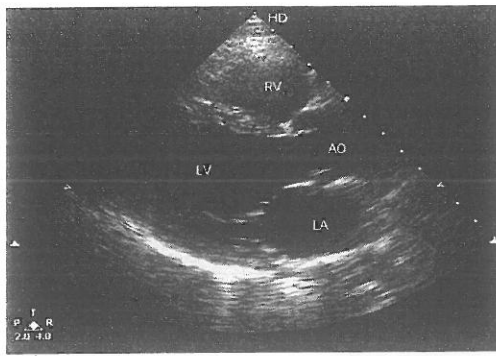
(2) Differentiate pulseless electrical activity from asystole (asystole by ultrasound = very poor prognosis)

(3) Detect pacer capture

b. Effusions: useful in medical as well as trauma patients

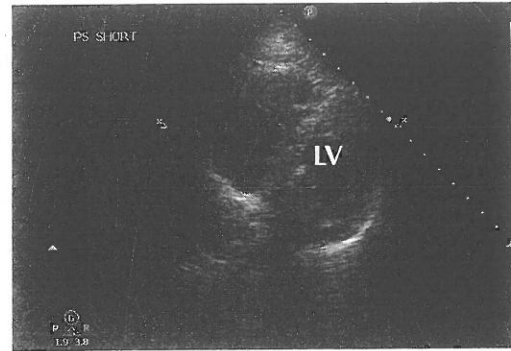
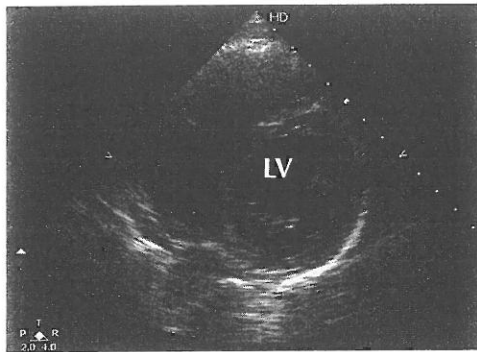
(1) Findings of tamponade: right ventricle collapse in diastole

(2) Ejection fraction: EPs shown to be able to differentiate normal, depressed, severely depressed global wall motion



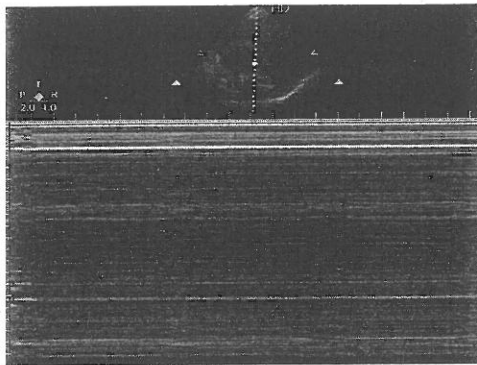
Courtesy of Sandra L. Werner, MD, RDMS, FACEP

The image on the left demonstrates a normal parasternal long axis view. The image on the right demonstrates a large effusion (arrows).



Courtesy of Sandra L. Werner, MD, RDMS, FACEP

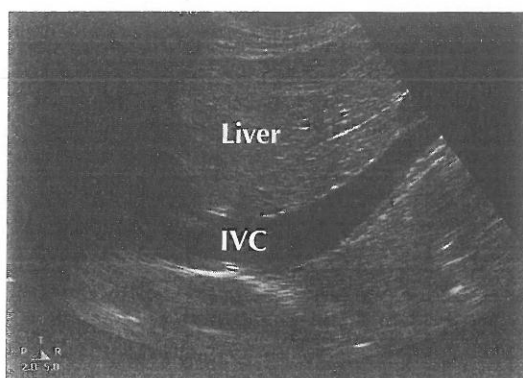
The image on the left is a normal parasternal short axis view. Note the round left ventricle (LV). The image on the right demonstrates right ventricular strain. Note the flattening of the septum, causing the left ventricle (LV) to look like a "D."



Courtesy of Sandra L. Werner, MD, RDMS, FACEP

Image demonstrating M-mode through the heart valves. No motion is seen, indicating asystole.

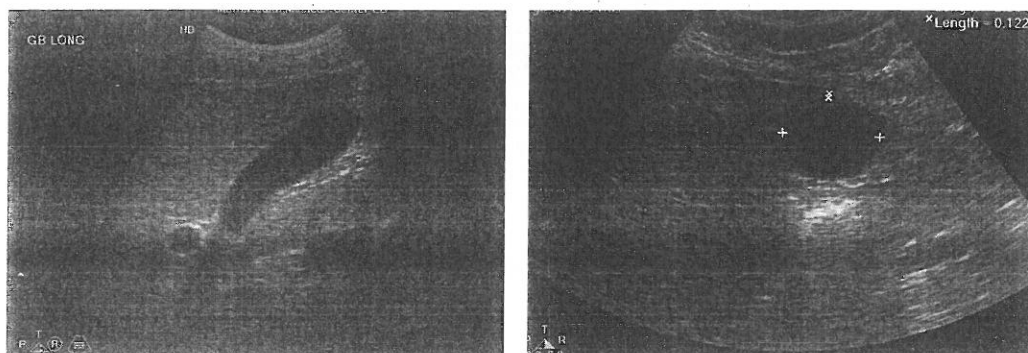
- c. Central pressures: IVC size and collapsibility assessed
 - (1) Small, collapsible IVC corresponds with hypovolemia.



Courtesy of Sandra L. Werner, MD, RDMS, FACEP
Image demonstrating the IVC in long axis

F. Biliary ultrasound

1. Goal: identify cholelithiasis, cholecystitis
2. Indications: right upper quadrant pain, epigastric pain, vomiting
3. Technique
 - a. Low-frequency abdominal transducer
 - b. Gallbladder scanned in short and long axis
 - c. Complete examination includes common bile duct measurements
4. Findings
 - a. Normal gallbladder: anechoic, diameter <4 cm in short axis, wall thickness <3 mm, CBD <6 mm (up to 1 cm in elderly and after cholecystectomy)
 - b. Cholelithiasis: mobile, shadowing, echogenic foci within the gallbladder
 - c. Cholecystitis: signs include dilated gallbladder, wall thickening, positive sonographic Murphy's sign (point of maximal tenderness to transducer pressure is over the gallbladder) but is a clinical diagnosis
 - d. Sludge: nonshadowing, mobile foci of varying echogenicity within gallbladder
 - e. Dilated CBD raises concern for choledocholithiasis
5. Clinical utility: fast and accurate with sensitivity of 94%, specificity of 96%

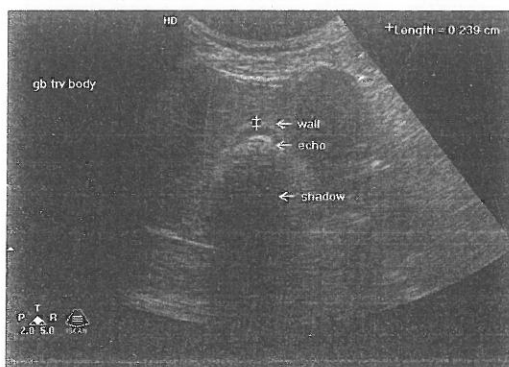


Courtesy of Sandra L. Werner, MD, RDMS, FACEP
Images of a normal gallbladder with wall and diameter measurements in the sagittal and transverse planes



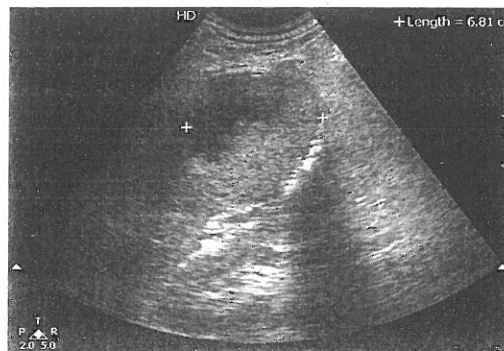
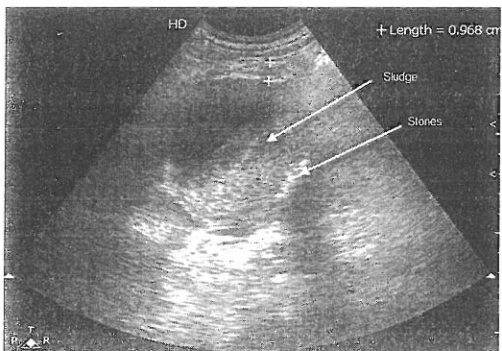
Courtesy of Sandra L. Werner, MD, RDMS, FACEP

This image demonstrates cholelithiasis without evidence of cholecystitis.



Courtesy of Sandra L. Werner, MD, RDMS, FACEP

This image demonstrates a gallbladder completely full of stones. The "wall, echo, shadow" (WES) sign is present.



Courtesy of Sandra L. Werner, MD, RDMS, FACEP

These images demonstrate findings of cholecystitis. In addition to the presence of stones and sludge, the gallbladder is distended and the wall thickened.

G. Renal ultrasound

1. Goals

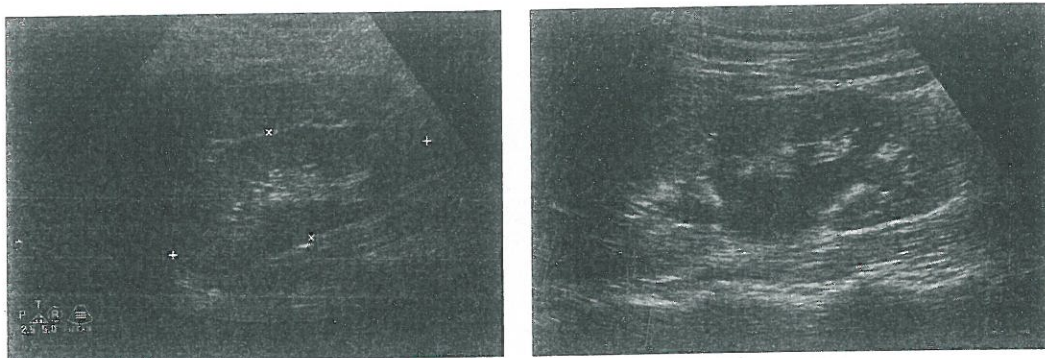
- Primary: identify hydronephrosis in the setting of suspected ureteral obstruction
- Secondary: measure bladder size to calculate post void residual and identify presence of urine before performing pediatric bladder catheterization

2. Technique

- Scan kidneys in sagittal plane (similar to FAST windows)
- Scan bladder in transverse and sagittal planes

3. Findings

- Normal kidneys: hyperechoic renal pelvis, hypoechoic parenchyma



Courtesy of Sandra L. Werner, MD, RDMS, FACEP

The image on the left demonstrates a normal kidney. The image on the right demonstrates hydronephrosis with a dilated collecting system.

- Hydronephrosis: dilated, anechoic renal pelvis, ureter may be dilated as well
- Ureteral stone: sometimes identified as echogenic, shadowing foci at UPJ or UVJ
- Bladder dimensions used to calculate volume

4. Clinical utility

- Reduced radiation exposure compared to CT
- Sensitivity of 83% and specificity of 92% for detecting hydronephrosis but not all renal stones cause obstruction
- Cannot definitively exclude renal colic with ultrasound

H. Deep-vein thrombosis ultrasound

1. Goal: identify vascular thrombus

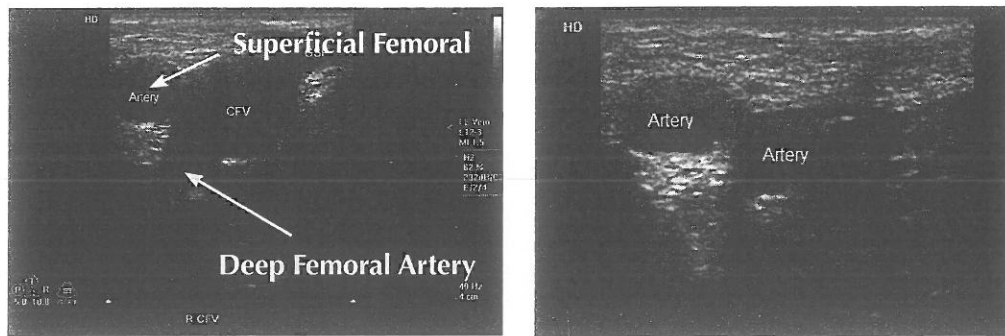
2. Indication: swollen lower extremity

3. Technique

- Usually high frequency transducer
- Compress from CFV through popliteal vein
 - Two point: CFV and popliteal only
 - Three point: adds junction of SFV and DFV

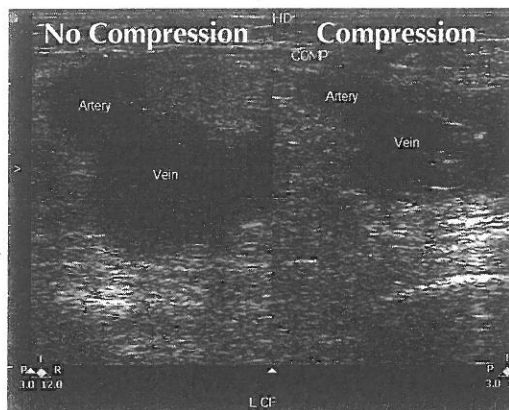
4. Findings

- Normal vein – anechoic, compresses completely



Courtesy of Sandra L. Werner, MD, RDMS, FACEP

The image on the left demonstrates a normal, noncompressed common femoral vein and the femoral arteries. The image on the right demonstrates normal complete compression of the common femoral vein.



Courtesy of Sandra L. Werner, MD, RDMS, FACEP

This image demonstrates a clot within the common femoral vein. The left image is without compression, and the right image is with compression. Notice on the right the vein is noncompressible and contains echogenic material.

5. Clinical utility

- a. Accuracy ranges from 70% to 99%, depending on operator experience.
- b. Emergency-physician performed studies shown to reduce emergency department length of stay and hospital charges

I. Procedural ultrasound uses

1. Vascular: peripheral and central lines, A-lines
2. Torso fluid: paracentesis, thoracentesis, pericardiocentesis, bladder
3. Cardiac: pacer placement
4. Musculoskeletal: arthrocentesis, fracture reduction, lumbar puncture landmarks
5. Soft tissue: foreign body removal, abscess versus cellulitis identification, abscess drainage
6. Anesthesia: peripheral nerve blocks
7. Airway: endotracheal tube placement
8. Ocular: retinal detachment, vitreous hemorrhage, increased ICP

PROCEDURES AND SKILLS: PRACTICE CLINICAL SCENARIOS

Answers immediately follow the procedures and skills practice clinical scenarios.

Scenario A

Presentation: A morbidly obese patient with CHF presents with pulmonary edema. He is started on nitroglycerin and given furosemide and placed on BiPAP. After 30 minutes on BiPAP, a blood gas demonstrates worsening acidosis and increased pCO₂.

What is the management?

Scenario B

Presentation: A middle-aged man collapses at a wedding and receives immediate CPR. EMS arrives and diagnoses ventricular fibrillation arrest. ROSC is achieved. The patient is intubated and transported to the emergency department.

What is the management?

Scenario C

Presentation: EMS responds to a reported gunshot wound. The patient is a 20-year-old man with a gunshot wound to the chest. There is a single, small wound on the anterior left chest wall. The patient is responsive on the arrival of EMS but quickly deteriorates. On arrival in the emergency department, EMS is ventilating the patient with a bag-valve-mask. Two large-bore IV lines have been established, and the paramedics report the patient had a pulse during transport. The patient has no vital signs in the emergency department.

What is the management?

Scenario D

Presentation: A 5-year-old boy falls from a jungle gym and sustains a colles fracture to the right wrist. He has mild asthma but no other injuries or medical problems. His last meal was with a milk shake 2 hours ago. The fracture requires reduction.

What is the management?

Scenario E

Presentation: A 40-year-old woman presents to the emergency department with 6 hours of right upper quadrant pain, nausea, and vomiting.

What is the management?

ANSWERS TO PRACTICE CLINICAL SCENARIOS

Scenario A

Management: Intubation is indicated for patients who do not respond to a trial of BiPAP. RSI may not be method of choice given obesity and baseline hypoxia. If RSI is attempted, a ramped position and preoxygenation are critical. Etomidate and propofol dosing is based on lean body weight, and succinylcholine on total body weight.

Scenario B

Management: The patient is a candidate for therapeutic hypothermia. Goal temperature is 33° as soon as possible, achieved by 2 L cold lactated Ringer's, ice packs to axilla and groin, cooling blankets. PTCA not contraindicated during cooling process for STEMI/evolving MI. Medications indicated during cooling include fentanyl or midazolam as sedation/seizure prevention, vecuronium if needed to prevent shivering (paralysis), and ASA per rectum. Additional measures include end-tidal CO₂ 35–40 mmHg, IV insulin if blood glucose >150 mg/dL, and transfusion if Hgb <10 mg/dL.

Scenario C

Management: Patients with the best survival after emergency department thoracotomy are those with penetrating chest trauma who arrest in the field. Emergency department thoracotomy is indicated for penetrating trauma when a trauma surgeon is available to take patient to operating room if resuscitated and there is at least one sign of life (blood pressure, pulse, respiratory effort, ECG rhythm, echo cardiac activity), or tamponade by ultrasound, or signs of life at scene and paramedic CPR <10 minutes.

Perform thoracotomy incision in fourth intercostal space on left; incise from right side of sternum to left posterior axillary line. Selectively ventilate right lung if possible. For cardiac massage, the two-hand technique is superior to one. The heart can be repaired with staples or sutures (3-0 silk). Can inflate Foley in wound, perform purse-string suture.

Scenario D

Management: There is no evidence to support the need to fast before sedation in children. A moderate level of sedation will be required. Ketamine is a good choice, because it provides dissociative sedation and analgesia. Vomiting is common after the procedure.

Scenario E

Management: Bedside biliary ultrasound is indicated. Ultrasound findings consistent with cholecystitis include stones, gallbladder wall thickening (>3 mm), gallbladder distension, positive sonographic Murphy sign, and pericholecystic fluid.

OTHER CORE COMPETENCIES OF THE PRACTICE OF EMERGENCY MEDICINE

EMERGENCY DEPARTMENT ADMINISTRATION	1175
General Statistics (United States)	1175
The Emergency Department Director	1175
Rules Governing Emergency Department Operation	1176
Record Keeping	1178
Risk Management.....	1179
Quality Assurance and Chart Audits.....	1180
ETHICAL-LEGAL ASPECTS OF EMERGENCY MEDICINE	1181
Consent to Treatment.....	1181
Statutes Affecting the Practice of Emergency Medicine	1186
Liability	1189
Medical Records	1193
Resuscitation Issues.....	1194
Miscellaneous Issues	1196
PHYSICIAN-PATIENT INTERACTION.....	1200
Attitudes of the Emergency Physician	1200
Traumatized Victims of Violence	1201
Families of the Critically Ill or Injured and Families of Patients Who Die.....	1202
The Hostile, Angry, or Uncooperative Patient	1203
The Substance Abuse Patient.....	1203
Patient Disposition	1204
PROFESSIONALISM.....	1208
Advocacy	1208
Diversity Awareness	1209
Ethical Principles.....	1209
Electronic Communications and Social Media	1211
Leadership and Management Principles.....	1212
Well-being	1212

OTHER CORE COMPETENCIES: SELF-ASSESSMENT QUESTIONS

1. Which of the following regarding consent is true?
 - (a) It can only be written and never implied.
 - (b) It may be given by a patient who has been declared by a judge to be incompetent.
 - (c) Consent for an unconscious patient may be obtained from the patient's family.
 - (d) It cannot be withdrawn by the patient once given.
2. In the process of obtaining an informed consent for a procedure, the physician must do all of the following except:
 - (a) Explain the procedure to be performed as well as its risks and benefits.
 - (b) Explain the risks and consequences of not having the procedure done.
 - (c) Inform the patient of alternative procedures as well as their risks and benefits.
 - (d) Advise the patient of the relative cost of the procedures.
3. Which of the following minors may not consent for their own treatment?
 - (a) Minors requesting treatment for life-threatening conditions that require immediate care
 - (b) Minors requesting treatment for a sexually transmitted infection
 - (c) Minors requesting treatment for a pregnancy-related complaint
 - (d) Minors requesting treatment for an injury that is not life threatening
4. Which of the following statements regarding Good Samaritan laws is most accurate?
 - (a) They are designed to protect people (particularly medical personnel) who respond to an emergency for which they are not being compensated.
 - (b) They are the same in each state.
 - (c) They provide liability protection for the provision of care by the responder, even if it is grossly negligent.
 - (d) They are directly applicable to the normal practice of emergency medicine by the emergency physician.
5. The four conditions that a plaintiff must prove to sustain a lawsuit for malpractice include all of the following except:
 - (a) Duty to treat
 - (b) Breach of duty
 - (c) Compensable injury
 - (d) Intentional wrong doing
 - (e) Proximate cause

6. Abandonment is most accurately described as:
 - (a) A breach of duty known as nonfeasance
 - (b) An intentional tort
 - (c) A breach of duty known as malfeasance
 - (d) A criminal tort
7. The manner in which an error in a medical chart should be corrected once the medical chart has been filed and the patient is no longer in the emergency department is by:
 - (a) Scribbling through the incorrect information so that it is no longer legible.
 - (b) Destroying the old chart and writing up a new one.
 - (c) Drawing a single line through the incorrect statement(s), noting the reason for the change, and initialing the correction.
 - (d) Adding a signed, dated, and timed addendum to the chart.
8. The Emergency Medical Treatment and Active Labor Act (EMTALA) applies to:
 - (a) Free-standing clinics
 - (b) Hospitals with a Medicare participation agreement with the government and an emergency department
 - (c) Nursing homes
 - (d) All of the above
9. Which is true regarding EMTALA?
 - (a) The patient does not need to be examined before transfer if he or she is unstable.
 - (b) The patient should be asked about ability to pay.
 - (c) The patient does not need to be transferred with a copy of the chart if he or she can personally give all the information to the new hospital.
 - (d) Patients who remain unstable despite resuscitative efforts can be transferred if the benefits of transfer outweigh the risks.
10. Violations of EMTALA can result in:
 - (a) Civil monetary penalties
 - (b) Termination or suspension of Medicare provider agreements
 - (c) Civil action
 - (d) All of the above
11. Which of the four elements of a negligence suit is most easily established for emergency department patients?
 - (a) Duty
 - (b) Breach of duty
 - (c) Causation
 - (d) Damages

12. All of the following are necessary in a *res ipsa loquitur* suit except:
- (a) The instrument causing the injury was in the physician's exclusive control.
 - (b) The patient did not contribute to the injury.
 - (c) The damages would not have occurred in the absence of negligence.
 - (d) There is direct evidence that the physician caused the damages.
13. What is the usual standard by which a plaintiff (patient) needs to prove negligence in a malpractice action?
- (a) Beyond a reasonable doubt
 - (b) Preponderance of evidence
 - (c) Obvious relationship
 - (d) None of the above
14. All of the following torts require intent except:
- (a) Battery
 - (b) False imprisonment
 - (c) Infliction of emotional distress
 - (d) Medical malpractice
15. Many states do not have mandatory reporting or immunity provisions for:
- (a) Child abuse
 - (b) Spouse abuse (domestic violence)
 - (c) Elder abuse
 - (d) All of the above
16. In a malpractice action the standard of care is:
- (a) Usually determined by a national standard
 - (b) Higher for specialists
 - (c) Usually determined by expert witness testimony
 - (d) All of the above
17. Most patients who are angry or physically threatening are:
- (a) Frightened
 - (b) Personality disorder with a history of violence
 - (c) Delirious or intoxicated
 - (d) Grieving

18. Which of the following is considered elder abuse?
- (a) A stranger striking a 90-year-old at the bus stop
 - (b) A son refusing to pick up his 90-year-old mother's mail daily instead of weekly
 - (c) A 90-year-old who lives alone who refuses to take her medication
 - (d) A son who uses his power of attorney to obtain credit cards in his mother's name for his personal use
19. What is the best predictor of how an angry patient will express his or her anger?
- (a) The patient's overall strength
 - (b) The patient's ethnic background
 - (c) The patient's past behavior
 - (d) The patient's sex
20. Which of the following is true about a living will?
- (a) It goes into effect only when a person is permanently unconscious or has a terminal condition.
 - (b) It can be used by prehospital providers to make decisions about providing oxygen to a patient via mask.
 - (c) It can be used by hospital staff to justify withholding pain medication.
 - (d) It can be used by a family to divide the assets of a deceased patient.
21. The most appropriate discharge disposition for an elderly patient who does not need hospital admission but requires daily medications, oxygen therapy, and close nursing supervision is:
- (a) A skilled-nursing facility
 - (b) An emergency shelter
 - (c) An adult foster-care home
 - (d) A room and board home
22. The state Department of Health mandates reporting all of the following except:
- (a) Cases of sexually transmitted disease
 - (b) Victims of gunshot and stab wounds
 - (c) Births that occur in the emergency department
 - (d) Victims of child or sexual abuse
23. On average, what percentage of hospital admissions comes from the emergency department?
- (a) 10%
 - (b) 20%
 - (c) 50%
 - (d) 80%

ANSWERS

- | | | | |
|------|-------|-------|-------|
| 1. c | 7. d | 13. b | 19. c |
| 2. d | 8. b | 14. d | 20. a |
| 3. d | 9. d | 15. b | 21. a |
| 4. a | 10. d | 16. d | 22. c |
| 5. d | 11. a | 17. c | 23. c |
| 6. a | 12. d | 18. d | |

Use the pre-chapter multiple choice question worksheet (page xx) to record and determine the percentage of correct answers for this chapter.

EMERGENCY DEPARTMENT ADMINISTRATION

I. GENERAL STATISTICS (UNITED STATES)

- A. 136.1 million visits each year
- B. 45.1 visits per 100 persons
- C. 21.7% of patients seen in <15 minutes
- D. 12.6% of visits result in hospital admission
- E. 2.0% of visits result in transfer to a different hospital (psychiatric or other)
- F. Characteristics of emergency department visits in 2010 among adults ≥ 18 years old
 - 1. 21.4% of people visited an emergency department one or more times
 - 2. Range 18.6% (patients 45–54 years old) to 27.4% (patients ≥ 75 years old)
 - 3. 18.5% of males and 24.3% of females visited an emergency department at least once.
- G. Visits to the emergency department in 2010 among adults 18–64 years old by type of insurance
 - 1. Private insurance 17.4%
 - 2. Medicaid 40.2%
 - 3. Uninsured 21.3%
- H. 50% admissions from the emergency department
- I. Admission rates vary across emergency departments from 9.8% to 25.8%.

II. THE EMERGENCY DEPARTMENT DIRECTOR

A. Qualifications

- 1. An emergency department director must be trained in emergency medicine, typically through a residency program.
- 2. He or she has demonstrated the following:
 - a. Dedication to quality emergency care
 - b. Leadership ability
 - c. Clinical competence
 - d. Administrative (management) skills
 - e. Teaching ability
- 3. An emergency department director must maintain continued clinical competence by practicing emergency medicine.

B. Responsibilities

- 1. Maintains day-to-day operations of the emergency department
- 2. Assures clinical competence of staff emergency physicians

3. Fulfills designated administrative duties
4. Determines work shifts and schedules emergency physician coverage
5. Represents the emergency department within the medical staff structure

III. RULES GOVERNING EMERGENCY DEPARTMENT OPERATION

A. The Joint Commission requirements (full hospital approval is for 3 years)

1. The Joint Commission is the largest of several hospital accrediting agencies and is a voluntary national accrediting body. Its mission is to enhance the quality of healthcare provided to the public. Loss of Joint Commission accreditation may engender sanctions of significant financial consequence, ie, the hospital may no longer contract with the federal government for Medicare reimbursement.
2. Any patient presenting to the emergency department must be evaluated.
 - a. If the patient is treated in the emergency department, he or she must be referred for follow-up care.
 - b. If no treatment is given, the patient should be advised and referred to an appropriate facility or service.
3. The emergency department must be directed by a physician who is an active member of the medical staff; in addition, the emergency department must be staffed appropriately and have available an appropriate range of consultants.
4. Ancillary services must be coordinated with the operation of the emergency department.
5. All emergency department personnel must be properly trained to deal with emergencies.
6. A comprehensive policies and procedures manual is compiled for the emergency department and should be regularly reviewed (and updated) to reflect the most current Joint Commission and institutional practice standards.
7. Medical records of all emergency department visits shall be maintained and become part of the permanent hospital records.
8. The emergency department must be accessible to the public and have an easily identifiable entrance. The emergency department must meet the minimum requirement for equipment, drugs, and supplies. There must also be a communications system and observation beds within the department.
9. An active quality review and quality control program must exist for the emergency department.
10. To comply with federal guidelines, the emergency department must provide language assistance to non-English-speaking patients. Do not use family members as interpreters; a closed-circuit TV with a live interpreter or a telephone translation service is preferred.

B. Emergency Medical Treatment and Labor Act (EMTALA)

1. Federal law that requires anyone coming to an emergency department to be stabilized and treated
2. Part of the Consolidated Omnibus Budget Reconciliation Act (COBRA) of 1985
3. The purpose is to prevent hospitals from transferring patients without a medical screening examination.
4. EMTALA violations are punishable with fines to hospitals and physicians.

5. Emergencies are defined by the law as "a condition manifesting itself by acute symptoms of sufficient severity (including severe pain) such that the absence of immediate medical attention could reasonably be expected to result in placing the individual's health in serious jeopardy." This includes a woman in labor.
6. EMTALA only applies when a patient presents to an emergency department and not an outpatient clinic that is not equipped to handle medical emergencies.
7. Under EMTALA, hospitals must provide a medical screening examination regardless of that patient's ability to pay, must post signs notifying patients and visitors of their rights to a medical screening examination, and must provide treatment for any emergent medical condition found until it is resolved or stabilized; if the hospital does not have the capability to treat the condition, an appropriate transfer must be done. In addition, a hospital with specialized capabilities must accept transfers from hospitals without those capabilities.
8. Under EMTALA, the patient must be categorized as stable or unstable by the treating physician. EMTALA does not apply to stable patients. For unstable patients, EMTALA states that there must be a documented medical benefit to transfer or the patient makes a transfer request in writing understanding the risks. The transferring hospital must care for the unstable patient until the transfer, must provide copies of the medical records, and must confirm the receiving facility has the capability to accept the transfer; the transfer must be made with appropriate personnel and equipment.
9. EMTALA is an unfunded mandate.

C. Policies and procedures

1. Developed by the emergency department director and approved by the hospital administration and medical staff, the policies and procedures provide guidance for clinical, regulatory, and administrative actions in the emergency department.
2. Departmental policies may be exclusive for the emergency department or shared among other departments, and may govern only the hospital-employed emergency department staff, or the staff plus emergency department physicians.
3. The Joint Commission and the Centers for Medicare & Medicaid Services often mandate consistent policies across the entire hospital (eg, conscious sedation).
4. Medical staff bylaws, rules, and regulations

D. Other governing agencies

1. State Department of Health
 - a. State rules and regulations supersede hospital/departmental policies.
 - b. Mandatory reportable cases (see also Ethical/Legal Aspects of Emergency Medicine, pages 1187–1189)
 - (1) Communicable diseases
 - (2) Victims of violence: state dependent (check local laws)
 - (a) Abuse (child, spouse, elder, or disabled)
 - (b) Alleged sexual assault
 - (c) Assault
 - (d) Gunshot wounds
 - (e) Stab wounds
 - (3) Dog bites: reporting policies vary by county and state
 - (4) Deaths (including dead-on-arrivals [DOAs] and abortions)

2. American College of Emergency Physicians (ACEP) requires 150 continuing medical education (CME) credit hours every 3 years (~50/year); 60 must be designated as ACEP category I credit.
3. American Board of Emergency Medicine (ABEM) requires recertification using continuous learning and intermittent testing to maintain board certification status.
 - a. Lifelong Learning and Self-Assessment: primary goal is to promote professional development by reading an annual set of articles chosen by ABEM and taking an open-book test and by completing 25 credits of CME per year on average. Eight of these must be self-assessment credits. Emergency physicians must pass four LLSA tests every 5 years.
 - b. Assessment of Practice Performance: The goal is to promote assessment of care compared with that of peers and apply best evidence to improve that care. Physicians must complete one practice improvement activity every 5 years (by collecting data on one aspect of care, reviewing data, and participating in a practice improvement plan) and one communication/professionalism activity every 5 years (by participating in patient experience of care surveys).
 - c. Continuous Certification (ConCert™) Examination: physicians must pass during the last 5 years of certification.
 - d. Maintaining professional standing: physicians must hold at least one medical license in the United States or Canada that is current, active, valid, and unrestricted.

IV. RECORD KEEPING

A. Emergency department record

1. There should be a durable emergency department record, either a hard copy that becomes part of the hospital medical record, or an electronic record.
2. The emergency department record must be legible; a dictated chart or use of templates (eg, T-system) or electronic medical records is preferable to handwritten.
3. Patient instructions should be specific, written or printed, and individualized as necessary. The instructions should be part of the medical record.
4. The confidentiality provisions of the Health Insurance Portability, Accessibility and Affordability Act (HIPAA) require that the emergency department record and protected health information be maintained at all times.

B. Emergency department log (a mandate of The Joint Commission)

1. The following information should be documented on all patients:
 - a. Time of arrival
 - b. Mode of arrival (ambulance, ambulatory)
 - c. Demographics (name, age)
 - d. Presenting complaint
 - e. Ancillary testing
 - f. Clinical impression
 - g. Room number (if admitted) or specialty unit (ICU, cardiac care unit)
 - h. Time of discharge

V. RISK MANAGEMENT

A. Patient charts (medical record)

1. Basis for defense in virtually every claim
2. Must be concise, complete, and legible
3. All information contained within is considered confidential

B. Malpractice risk is closely related to patient satisfaction; patients tend not to sue physicians they like.

C. Documentation

1. The patient's complaint should be recorded in his or her own words. Absence of a specific complaint should also be noted. For example: "Patient states he doesn't feel well. He has no specific complaint."
2. Initial vital signs must be clearly visible on the chart and periodically rechecked (if appropriate) and recorded.
3. All findings from the following sources must be recorded.
 - a. History and physical examination, which should include all positive (and significant negative) findings. For example: "The patient complains of shortness of breath and nausea but denies having chest pain."
 - b. Diagnostic evaluation
 - c. Consultation
 - d. Disposition
4. The exact discharge instructions must be recorded including follow-up care for all patients.
5. Consent for treatment and all procedures must be documented on the chart.
6. Writing in-patient orders may extend the emergency physician's liability risk into the hospital. This practice is to be discouraged. In the event that it is necessary for the patient's well-being, include the following information on the order sheet:
 - a. The orders are written on behalf of the patient's attending physician (include his or her name).
 - b. The attending physician should be notified immediately of changes in the patient's condition.

D. Types of consent

1. Expressed consent: a verbal or written expression of willingness to be treated (includes all "usual" treatments)
2. Implied consent: by his or her action, the patient implies willingness to be treated (eg, patient rolls up sleeve for an injection).
3. Informed consent: patient is informed of the potential risks and benefits of the procedure, any alternative procedures, and the consequences of not having the procedure before giving his or her consent. (For more details, see consent to treatment in Ethical/Legal Aspects of Emergency Medicine, pages 1181–1186.)

VI. QUALITY ASSURANCE AND CHART AUDITS

- A. Types of chart reviews (audits): note that The Joint Commission and Medicare are moving toward outcome-driven peer review by provider.
 - 1. Random review of daily charts
 - 2. Physician review (directed toward a specific physician)
 - 3. Complaint-oriented review (charts of selected presenting complaints, ie, chief complaints)
 - 4. Monthly death reviews (charts of patients who died in the emergency department or within 24 hours of hospital admission)
 - 5. Chart review of patient who returns within 72 hours
- B. Quality assurance process
 - 1. Identification of cases in which quality assurance criteria have not been met
 - 2. Review of charts by an emergency physician and hospital audit or quality assurance committee
 - 3. Feedback to the health care provider who deviated from the criteria
 - 4. Ongoing review of the healthcare provider's charts

ETHICAL-LEGAL ASPECTS OF EMERGENCY MEDICINE

I. CONSENT TO TREATMENT

A. Expressed consent

1. May be verbal, written, or both, and is obtained from patients who are alert and mentally competent
 - a. All adult patients are presumed competent to consent to or refuse care.
 - b. The problem with oral consent is proving it in court; hence, written consent is preferred whenever possible.
2. Consent must be given free of suggestion of duress or coercion. There are times when a patient seeks care when a third party, a friend, or an employer, or a police officer has suggested it. The physician must ensure the patient is freely giving consent to treat; otherwise, there may be no consent at all.
3. General consent to treatment: patient requests emergency department care (verbal) and signs a general consent form (written), affirming his or her willingness to receive general care.
4. Informed consent for an invasive or risky procedure or treatment
 - a. Informed consent: the patient must be given adequate explanation about the procedure or treatment, its outcomes, risks, and alternatives.
 - b. The obligation to obtain consent rests with the physician who is going to perform the procedure or treatment.
 - c. Verbal informed consent process: For example, the patient is told that a specific invasive procedure (eg, peritoneal lavage) is required to make a diagnosis. The risks and benefits are explained, the patient is informed of any alternative procedure (ultrasound) and its potential consequences or outcome; the patient is also made aware of the consequences (eg, intraperitoneal bleeding) of not having any diagnostic procedure performed. The patient is then asked if he or she understands this information and if he or she agrees to the procedure. A "yes" answer is a verbal informed consent and should be obtained and charted as such by the physician performing the procedure.
 - d. Written informed consent process: For example (continued), the nurse again asks the patient if he or she understands what the information provided by the emergency physician and if he or she agrees to the procedure. If a "yes" answer, the nurse asks the patient to sign an informed consent form to affirm his or her understanding of and willingness to undergo the procedure.
 - e. Failure to secure an informed consent for an invasive procedure may allow the court to find that a battery (unconsented intentional touching) has occurred. The court may also find the emergency physician guilty of false imprisonment (unlawful detention or restraint of an individual's personal liberty or freedom). Battery and false imprisonment are good examples of intentional torts in which it is not necessary to prove negligence (which is the usual basis for a malpractice lawsuit); instead, the patient (plaintiff) needs to prove intent.
 - f. An informed consent may be withdrawn by the patient at any time and may be oral or written; however, written consent is preferred.

- g. What is considered in one jurisdiction to be an invasive procedure that requires more in-depth informed consent may not be considered to be invasive in another.
- h. The consent form itself is not the consent. The form is a written confirmation that the conversation about consenting for the procedure or treatment has occurred and that the patient agrees.
- i. Consent forms may have a place for a witness to sign. This signature confirms the identity of the person giving consent and not the adequacy of the consent process (eg, the surgeon is skilled in the procedure, or the explanation was good).
- j. A signed informed consent in the record is important, but a physician note of the discussion may be of equal or greater usefulness for defense purposes.

B. Implied consent

- 1. Inferred when a patient conveys consent to treatment by his or her actions or when he or she is not alert or mentally competent to give consent in an emergency situation
- 2. Implied consent by action
 - a. Passive implied consent (eg, lack of resistance to injection or phlebotomy)
 - b. Active implied consent (eg, rolling up sleeve for injection or phlebotomy)
- 3. Implied consent by condition (substituted judgment doctrine)
 - a. In a patient with an altered level of consciousness or one who is determined to be mentally incompetent, this doctrine is based on the assumption that "the patient would agree if he or she could."
 - b. This form of consent is valid if treatment is necessary to prevent loss of life or health and, in some jurisdictions, is referred to as the emergency doctrine. The emergency physician has consent (as a matter of law in these cases) to carry out any assistance reasonably required to stabilize the patient's condition until consent can be obtained.
- 4. Presentation to the emergency department with a chief complaint might imply consent for a history and physical examination. However, invasive examinations such as pelvic or rectal examinations, or examination of the genitals or breasts, should be preceded by the physician stating that such an examination will be done and obtaining verbal consent.

C. Competence to render consent

- 1. Minors and mentally incompetent persons are presumed to be incapable of rendering legally binding consent.
 - a. Because minors lack legal competence to consent for treatment, authorization to treat is often required from a parent or legal guardian under state law. However, EMTALA preempts state law and requires the hospital/emergency department to examine all minors presenting to the emergency department to determine if an emergency medical condition is present. If so, EMTALA also requires the hospital and emergency department to stabilize the emergency medical condition. Examination and stabilization must be done without delaying to obtain consent from the minor's parent or legal guardian.
 - b. If unable to contact a parent or guardian, document your efforts and decision to treat the problem whenever the condition would likely worsen without treatment. It is prudent to use the least invasive procedure available that is likely to achieve a satisfactory result.
 - (1) If circumstances allow, consider obtaining another physician's opinion before starting therapy.
 - (2) When there is an option, use the least invasive procedure or therapy consistent with good medical practice.

- c. A parent or legal guardian must always be sought to procure consent when an illness or injury is not life threatening.
 - d. If a parent refuses consent, and failure to treat would likely result in loss of life or health, what you do next is determined by how quickly you must intervene:
 - (1) A child of a Jehovah's Witness with life-threatening hemorrhage requires early intervention; try to get a second physician's opinion before starting therapy.
 - (2) If the same child has bleeding from injuries that are not life or limb threatening, seek court authorization to start therapy.
 - (3) In some jurisdictions, in cases involving a pregnant Jehovah's Witness, the courts have allowed emergency physicians to proceed with therapy, despite non-consent of the patient when the life of the fetus is at risk. The courts have ruled that the patient may "make a martyr of themselves, but not their children."
 - e. Failure to give "standard" medical care to a minor who is suffering from an emergency medical condition constitutes negligence.
2. An emancipated minor is an exceptional case, because the parents have entirely surrendered the right to treatment, custody, and earnings of the minor child. Some states (eg, Ohio) do not recognize minors as emancipated until they reach 18 years of age, even if they are considered emancipated in another state. Be sure you know your state's laws. Evidence of emancipation:
- a. An address separate from parents
 - b. A high school graduate
 - c. A married minor
 - d. A minor with dependent children
 - e. Self-supporting
 - f. In the military
 - g. Age
 - (1) As the chronological age of the patient approaches the statutory age of majority, some jurisdictions have held that the patient is emancipated (also referred to as the mature minor doctrine).
 - (2) When invoking this doctrine, the emergency physician should carefully evaluate and document the patient's developmental state and maturity in making these decisions.
3. All states have a statutory exemption allowing minors to consent to treatment for sexually transmitted infections, mental health, drug abuse, pregnancy-related care, and in some cases pregnancy prevention. Some states require the consent of a parent or guardian for pregnancy termination.
4. A "mentally incompetent" patient is one who is intoxicated (alcohol or other drugs), psychotic, confused, disoriented, or unconscious.
- a. Documentation of behavior, neurologic status, and psychiatric evaluation must be complete to support the conclusion of mental incompetence.
 - b. Next of kin, guardian, or power of attorney should always be consulted when available.
 - c. The substituted judgment doctrine may be applied in this setting. Examples:
 - (1) The intoxicated patient with a skull fracture
 - (2) The unconscious patient requiring a lumbar puncture to exclude meningitis
 - d. Because the intoxicated patient cannot render or withhold consent for treatment, he or she should be treated as needed and (if not admitted) observed until unimpaired.

5. Unique circumstances arise when minors are parents themselves. This may be state dependent, but in some circumstances the minor can give consent to treat his or her child but cannot give consent to treat him or herself.
6. Although minors cannot consent for most treatments or evaluations themselves, they should be part of the consent and decision-making process, depending on their age and maturity.
7. Patients who have a guardian or substitute decision maker, who have been committed, or who are under police custody may still be able to give consent for some medical procedures or examinations but not have the capacity to understand others (eg, a patient with mild dementia may be able to consent for a chest radiograph but not for surgery). This must be dealt with on a case-by-case basis. (See also capacity versus competency, page 1185.)

D. Refusal to give consent

1. Adult patients are always presumed competent and a competent, conscious adult has the right to refuse consent to all medical care, including care of a life-threatening condition. Supreme Court decisions have suggested a competent adult patient has a right to refuse life-sustaining or life-prolonging medical care. Again this "right to die" may be tempered by the courts when foreseeable third parties (single-parent patient with dependent young children, pregnant condition of patient, etc) are involved.
2. When a competent patient refuses treatment, careful and complete chart documentation is absolutely essential.
 - a. The emergency physician must document that the patient was told the reasons why he should remain for evaluation and treatment, what the evaluation and treatment consist of, what alternatives (if any) are available, and what risks versus benefits are associated with each scenario. In particular, it is imperative the patient be specifically informed of the worst case scenario (which is usually death).
 - b. A narrative record of the patient's refusal (his own words) should be noted in the chart.
 - c. The prudent emergency physician should also:
 - (1) Inform the patient who refuses treatment against medical advice (AMA) that he or she is welcome to return to the emergency department at any time if he changes his or her mind and agrees to be treated
 - (2) Ask the patient if he or she has any questions about what was discussed
 - (3) Inform the patient of his or her federal right to stabilizing treatment under EMTALA
 - (4) Provide treatment within the scope allowed by the patient, including antibiotics or pain medications as indicated
 - d. All of the above should be witnessed, if possible, by a nurse (or other health care provider) and a member of the patient's family (when present).
 - e. An AMA form should be completed in addition to the aforementioned chart documentation; however, a personal chart entry by the emergency physician (as described above) is more likely to support the contention that the AMA patient was fully informed of all possibilities by a physician before he left AMA.
3. Important considerations
 - a. A patient under arrest usually has the right to refuse treatment. However, in some states, the chief medical officer of the prison may consent for treatment (even though the prisoner refuses) if the injury was intentionally and willfully self-inflicted.
 - b. A patient expecting to see his or her private physician in the emergency department has the right to refuse treatment by the emergency physician. However, if the patient's

condition requires emergency intervention, the emergency physician should see him or her and provide appropriate care.

- c. In a patient who is mentally competent, blood tests for toxicology levels can be drawn without consent only if:
 - (1) There is a court order or
 - (2) It is authorized by a local statute, eg, in a negligent homicide setting.
- d. See also the section on collection of medical evidence, pages 1197–1199.

E. Parental refusal of medical care for a child

1. In general, most federal and state statutes uphold parental control over medical matters affecting their children. However, the courts have decided against parental wishes if the child's health is in jeopardy because of the parents' lack of consent. This is usually done under the doctrine of "parens patrie," which is the state's paternal interest in children.
 - a. Parents do not have the authority to forbid saving their child's life.
 - b. The fact that a parental refusal is based on claims of religious freedom does not change the court's position. The courts have held that denying medical care to a child is not within the parent's first amendment right of freedom of religion. "The right to practice religion freely does not include the liberty to expose a child to ill health or death."
2. If the parents withhold consent (and there is a life threat), the emergency physician should take temporary protective custody of the child, provide care as needed, admit the child based on the state's child abuse statutes, and contact the appropriate agencies (ie, local child protective agency and the hospital attorney).
3. If there is no immediate life threat, the parents' refusal of medical care should be respected, but this may be considered child neglect (not abuse). In this case, the emergency physician and Social Services could obtain a court order forcing the parents to permit initiation of specifically indicated treatment of the minor child.

F. Competency versus capacity

1. Capacity is a clinical determination of the ability to make medical decisions.
 - a. A physician must make sure a patient making a medical decision has the capacity to do so for each medical decision.
 - b. The patient must be able to communicate a choice, understand relevant information, appreciate the situation and its consequences, and reason about treatment options.
 - c. There are additional tools to help a physician determine if a patient has capacity to make a decision.
 - (1) Use follow-up questions or ask the patient to explain back the procedure or the treatment options.
 - (2) Use an additional test such as the Mini-Mental Status Examination or the MacArthur Competence Assessment Tool for Treatment (both of these tests are time consuming and may not be able to be done in the emergency department).
 - d. Patients with some impairment may be able to reasonably consent to some examination and treatments but may not be able to understand more risky or complicated treatment plans or choices. For example, a patient with mild dementia may be able to understand getting antibiotics for cellulitis but might not understand treatment choices for breast cancer.
2. Competency is a legal determination. A person is incompetent if unable to:
 - a. Respond intelligently to questions about treatment

- b. Participate in decisions with a rational thought process
- c. Understand basic information with respect to treatment
- 3. To assess competency, a court will assess mental alertness, orientation, ability to attend and concentrate, short- and long-term memory, ability to communicate, recognition of familiar objects, ability to understand quantities, ability to reason with abstract concepts logically, ability to carry out actions in one's own self-interest, and ability to modulate mood and affect. The person must also be free of uncontrollable intrusive thoughts and delusions.

II. STATUTES AFFECTING THE PRACTICE OF EMERGENCY MEDICINE

A. Mental health laws: emergency physicians generally have the power to commit patients who, as a result of mental illness, are deemed dangerous to themselves and/or others.

1. Common law provides the legal premise for commitment under the doctrine of "police power." This allows the state to ensure the safety of its citizens.
2. For the emergency physician, the first step in the evaluation is to determine if the patient is a danger to himself (suicidal) or to others (homicidal or assaultive).
 - a. This may be facilitated by
 - (1) A history of past violent behavior or
 - (2) Statements by the patient as to his malevolent intentions
 - b. If the patient is indeed a danger to himself/herself or others, and has a mental disorder, he or she can be admitted/committed against his or her will and held for a fixed period of time (generally 48–72 hours).
 - c. If necessary, physical and/or chemical restraints may be used to be sure that a dangerous patient does not leave the emergency department.
 - d. Again, documentation is important. To avoid potential charges of false imprisonment or assault and battery at a later date, carefully document why you feel that the patient is dangerous (to himself/herself or others) and needs to be committed against his or her will.
3. Failure to commit
 - a. A number of successful malpractice actions have been brought against emergency physicians for failure to recognize suicidal risk and commit a patient.
 - b. "Failure to prevent suicide" is responsible for the highest average indemnity in emergency medicine malpractice cases.
 - c. Failure to commit a patient who poses a threat to known and foreseeable third parties is also associated with legal risk.
4. Voluntary admission
 - a. The suicidal or homicidal patient who initially agrees to voluntary admission may later change his or her mind and may then be free to leave the institution at will (state laws vary).
 - b. Therefore, any suicidal or homicidal patient transferred should be done so under involuntary admission or some type of hold papers valid under state law.

B. Good Samaritan laws

1. These laws are state-specific and are designed to protect only noncompensated responders to an emergency. Ordinarily, these statutes do not apply to the emergency physician engaged in the routine practice of emergency medicine.
2. A Good Samaritan law merely provides a legal defense to a responder's actions; it does not prevent a lawsuit (anyone can sue). However, it is extraordinarily rare for a physician to be successfully sued for treating a person in need of emergency care.
3. There is a standard of care in Good Samaritan cases. It is always a lower standard than ordinary negligence and is often statutorily stated that "acts or omissions intentionally designed to harm or any grossly negligent (or reckless) acts or omissions are not provided Good Samaritan protection." Therefore, if a responder's conduct is grossly negligent under the circumstances, there is no protection from legal liability.

C. Reportable conditions

1. Physicians are required to report certain conditions to the proper authorities. Failure to report may invoke criminal penalties or may be considered a deviation from a civil standard of care, ie, physician knows the condition is reportable but does not report it.
2. Some state-to-state variability in the types of conditions that must be reported exists; be sure you know the mandatory reportable conditions for your state.
3. Reportable conditions may include:
 - a. Sexual assault
 - b. Statutory rape (intercourse with female under statutory age): age depends on local laws (eg, a 16-year-old may consent to intercourse with a person of any age; a 13-, 14- or 15-year-old may consent to intercourse with a person within 2 years of his or her own age; and those ≤ 12 years old may not consent to intercourse with anyone regardless of age). Also note that state laws may consider it to be sexual misconduct or rape regardless of age if it occurs with a teacher or coach before the person graduates high school.
 - c. Abortion
 - d. Animal bites
 - e. Assault, battery, or other violent acts (such as stabbings or shootings)
 - f. Abuse (child, elderly, disabled): elder abuse is state dependent and may be limited to those older adults who are dependent on another for care or finances. For example, a 70-year-old woman who is punched in the face by her husband but is not seriously injured, unless she is dependent on him for care, may not be mandatory reporting in that state; in fact, it may be a violation of her privacy if she requests that you do not inform the police.
 - g. Neglect (defined as abuse without physical injury and includes the withholding or denying of nourishment or medical care)
 - h. DOAs and any death in the emergency department
 - i. Sexually transmitted infections and other communicable diseases (eg, hepatitis, pertussis)
 - j. Poisoning such as botulism
 - k. Motor vehicle collisions and illegal blood alcohol levels (in some states)
 - l. Concealed weapons (in some states)
 - m. Suspected biological incident (eg, anthrax)

4. Specific issues

a. Legal management of abuse/neglect

- (1) Physical abuse or negligent care of children should be reported to Protective Services in most states (another designated agency is used in some states).
 - (a) Breach of this standard of care may be considered not only negligent but also criminal.
 - (b) A physician can be charged retroactively if the patient sustains future battering and it is determined that a prior incident was not reported.
- (2) Minors who are at immediate risk of harm must be protected by removing them at once from the threatening environment.
 - (a) Hold minor patients in the emergency department until proper authorities arrive, or
 - (b) Admit the patient for further evaluation and investigation.
- (3) The parents' or guardians' rights do not overshadow the patient's right not to be beaten. If abuse is suspected, the decision to report the incident or admit the patient who is at immediate risk of harm is required even in the face of parental or guardian disapproval.
 - (a) Communications regarding abuse or neglect occurring between the emergency physician and a patient's parents or guardian are not privileged (protected).
 - (b) Most state abuse and neglect laws contain provisions whereby mandatory reporters are immune from civil and criminal liability.

b. Communicable diseases

- (1) The emergency physician's duty extends beyond merely reporting communicable disease cases to include:
 - (a) Warning the patient against engaging in activities that may spread the disease
 - (b) Informing the patient to notify contacts that may have been exposed
 - (c) When informing a patient that they have a communicable disease, it is important to know the laws that govern that patient's future behavior. In some states, it is a felony for those with HIV to have sexual intercourse without telling the sexual partner of their HIV status.
- (2) These efforts should be carefully documented to protect the patient, the contacts, and the treating physician.

c. Assault regulations

- (1) Wounds or other injuries that are known to be the result of a violent incident must be reported to the police. (The type of wounds and extent of injuries that require reporting are state dependent.)
- (2) Wounds or other injuries that raise suspicion of foul play should also be reported. (Know your state laws.)
 - (a) Any gunshot wound or major stab wound
 - (b) Any injuries resulting from an object that may have been used in a violent manner (eg, a baseball bat)
 - (c) Injuries that result in serious injury (eg, intracranial bleed, long-bone fracture)

d. DOA regulations

- (1) DOA cases should be reported to the coroner or medical examiner for possible investigation of foul play and to determine the need for postmortem examination.

- (2) Handling of the body should be minimized, and nothing should be done to alter the appearance of the corpse, because it may interfere with or complicate the evidence-gathering function of the coroner or medical examiner.

III. LIABILITY

A. Negligence (the predominant theory of liability in medical malpractice)

1. Definition

- a. Conduct other than that which a reasonable and prudent physician would or would not do under the same or similar circumstances
- b. It is a statewide (or nationwide) standard for the protection of others from unreasonable risk of harm.
- c. An adverse outcome is not synonymous with negligence, ie, a bad result is not necessarily a basis for a lawsuit.

2. Elements of negligence: a malpractice suit has four components: duty, breach of duty, causation, and damages. To succeed in court, the plaintiff (patient) must establish proof of all four components by a preponderance of evidence. This means the plaintiff need only establish that there is slightly more evidence in his favor (referred to as the 51% rule) than in favor of the defendant to succeed.

a. Duty to treat

- (1) Although physicians do not generally have a duty to treat anyone unless a professional relationship exists, the EMTALA imposes a legal duty on all Medicare-participating hospitals to render emergency care. Hence, a physician-patient relationship (or a hospital-patient relationship) for emergency medicine is established when a patient presents requesting medical care.
- (2) The duty imposed is that of treatment according to the prevailing standard of care.
 - (a) Standard of care is defined as those actions a reasonably competent physician would take under similar circumstances. The physician is not held to exercise the highest degree of skill and care ordinarily exercised by physicians within that same specialty, but rather that of a reasonably competent physician in that specialty.
 - (b) Standard of care is a state or national standard.
 - (c) Standard of care is higher for a specialist (such as an emergency physician) than for a generalist. Because emergency medicine is a specialty, nonemergency physicians practicing medicine in the emergency department will be held to the same standard as an emergency physician.
 - (d) Some standards are statutorily defined, ie, they conform to a written law (eg, abuse and reporting statutes). In this case, standard of care presumes that a competent physician knows and will abide by the law.
 - (e) Standard of care is otherwise usually determined via expert physician testimony at deposition and/or trial.
- (3) Example of duty to treat: If a patient comes into the emergency department and requests care, a physician-patient relationship is established. This places an obligation on the emergency physician to treat the patient according to the accepted standard of care. This duty is also based on EMTALA.

b. Breach of duty**(1) Types of breach**

- (a) **Malfeasance:** performing an action that should not have been done (eg, amputation for a simple finger laceration)
- (b) **Misfeasance:** performing an action in an improper (substandard) way (eg, suturing a laceration without conducting a thorough search for a foreign body)
- (c) **Nonfeasance:** failure to perform an action required by the circumstances (eg, neglecting to do a lumbar puncture in a child with meningismus)
- (d) **Failure to diagnose** (eg, diagnosing peptic ulcer disease in a patient who actually has an acute coronary syndrome)
- (e) **Failure to diagnose in a timely manner** (eg, allowing excessive time to pass before an ECG is done on a patient with chest pain)

(2) Abandonment

- (a) This is a type of nonfeasance in which the physician inappropriately terminates the physician–patient relationship.
- (b) Examples of abandonment in the emergency department setting
 - i. Admission of a patient to the care of a physician who does not see the patient before a catastrophe occurs; the admitting physician may be deemed to have abandoned a continuing duty to treat.
 - ii. Transfer of a patient to another hospital without evidence of physician-to-physician communication, including an agreement to accept the patient.
 - iii. Telephone orders given to a nurse by a primary care physician for treatment of an emergency department patient in lieu of seeing the patient; the emergency physician may be seen as having abandoned a duty to treat.

c. Causation (proximate cause)

- (1) This is often the most difficult of the four elements to establish.
- (2) A bad result without proof of causation does not constitute negligence.
- (3) Causation consists of two elements: causation in fact and foreseeability. The plaintiff must establish both.
 - (a) **Causation in fact:** This element states that “but for” the actions of the defendant (emergency physician), the injury would not have occurred.
 - (b) **Foreseeability:** The plaintiff’s damages must be the foreseeable result of the defendant’s breach in the standard of care.
- (4) In most cases, causation is established by expert testimony.
- (5) The proof of causation is determined by a “preponderance of evidence” standard, which means that the patient (in most cases) must prove that it was more likely than not that the emergency physician’s conduct caused the alleged harm. The court asks, “If the patient (plaintiff) was injured, considering all the possible causes, is it more likely than not that the physician’s act caused the injury?”
- (6) Although the “but for” test of causation is used by most jurisdictions, there are two other bases on which causation may be established.
 - (a) **Substantial factor:** This test of causation is particularly useful in cases involving multiple negligent acts. Using this approach, causation can be established even if less than a 50% chance of causation can be shown. In other words, even if other factors were involved in causing the damage, causation can still be established if the negligent action was a substantial factor in the damage.

- (b) **Loss of chance:** In some states (eg, Illinois and Pennsylvania), the patient can prove causation if he or she can establish that he or she was denied a chance of recovery/survival despite the fact that there was a less than probable chance of survival to begin with.
- (7) ***Res ipsa loquitur*** ("The thing speaks for itself") is a legal concept used when the circumstances of the case make it impossible to prove all the elements of negligence and shifts the burden of proof from the patient to the physician, who must prove that he or she was not negligent. The requirements for invoking this concept are:
 - (a) That the instrument causing the injury was in the physician's exclusive control;
 - (b) That the patient did not do anything that could have contributed to the injury; and
 - (c) The patient must prove that the damages would not have occurred in the absence of negligence. (Example: A patient awakens in the recovery room after an appendectomy with new onset paraplegia; negligence has occurred, but it is impossible to discover who was negligent and how the damages arose.)
- d. **Compensable injury (damages)**
 - (1) To be compensable, an obvious tangible injury must have occurred and must be the result of a breach in the standard of care. Allegations by the patient that a breach in the standard of care resulted in a divorce, or other such intangible results, is insufficient to win a medical suit.
 - (2) Theoretically, compensation is awarded for the losses caused by the injury, not the actual injury itself.
 - (3) Both general and specific damages may be collected by the winning plaintiff. These damages are typically expressed in pecuniary (monetary) form and are usually covered by malpractice insurance policies.
 - (a) **General damages**
 - i. Are the direct and proximate result of the injury
 - ii. They include:
 - Physical disability
 - Pain and suffering
 - Mental impairment
 - Loss of consortium
 - Loss of enjoyment of life
 - (b) **Specific damages**
 - i. Those that actually occur but do not generally and inevitably occur as a result of a particular injury.
 - ii. They include:
 - Loss of income
 - Medical expenses (present and future)
 - Costs of special equipment for care
 - (4) **Punitive damages** constitute a special type of compensable injury designed to set an example.
 - (a) They are awarded when the conduct of the emergency physician was egregious or showed reckless indifference to the rights or well-being of others

(eg, allowing an ill patient to lie unattended on an emergency department floor in the mistaken belief that he was malingering).

- (b) Punitive damages are not covered by malpractice insurance and are rarely awarded in malpractice cases.

B. Intentional torts

1. A separate entity from "negligence": differ from negligence torts (which form the basis of malpractice actions) in three ways.
 - a. No expert witnesses are necessary, because the standard of care is not an issue.
 - b. No actual or physical injury is needed.
 - c. Proximate cause is not required; in other words, injuries caused by the intentional tort need not be related to the tort itself.
2. Intentional torts pertinent to emergency department practice
 - a. Assault
 - (1) Placing a patient in fear of offensive touching
 - (2) Example: threatening to restrain a nonconsenting, competent patient
 - b. Battery
 - (1) Unconsented and intentional touching
 - (2) Example: performing an invasive procedure such as a lumbar puncture without consent
 - (3) Valid consent effectively bars this complaint.
 - c. False imprisonment
 - (1) Unconsented and unintentional confinement; this does not have to be physical or chemical restraint per se, but rather can occur merely by having a security officer stand by the patient's bedside.
 - (2) Example: the act of restraining a nonconsenting, competent patient
 - (3) Conditions under which you may restrain a patient
 - (a) Implied consent (eg, restraining an unconscious, confused or disoriented patient to save life or limb)
 - (b) Self-defense or defense of others (self-protection)
 - i. The state allows restraint for the patient's welfare and/or that of the community.
 - ii. Whenever restraints are placed, complete documentation detailing the need for restraint is mandatory and must be updated if the patient's condition changes.
 - iii. State laws, Joint Commission standards, and hospital policies exist for documenting the need for restraints, the continued observation of the patient, the repeated assessment of the restraints and the restrained patient (breathing, restraints causing chaffing), and the time until another assessment for the need for continued use of restraints.
 - d. Infliction of emotional distress or outrage
 - (1) Assumes the individual's right to be free from mental assault
 - (2) Example: misidentifying a patient and notifying the wrong family with resultant emotional damage

C. Privacy and confidentiality

1. Although patients themselves have a right to access their own medical records, communications with patients and written medical records are privileged and private.
 - a. Police do not have a right to a patient's medical information.
 - b. Release of such privileged information without the patient's consent is an invasion of privacy and is a compensable injury.
 - c. Consent should be obtained from the patient in the form of a signed release. Information may also be released with a court order.
2. Exceptions to the right of privacy and confidentiality
 - a. Minors and the mentally disabled: a parent or guardian is substituted as the party who can waive the privilege.
 - b. Statutorily required reporting: reportable conditions and disabled drivers
 - c. Public safety threat: if a patient tells a physician that he is going to harm another individual, and he names the victim, the physician has a duty to inform the police.
3. An absolute physician-patient privilege paralleling that which exists between an attorney and his client does not exist in court cases.

D. Joint and several liability

1. According to this concept, when two or more physicians are responsible for an act of negligence, all are held liable for damages and the patient can, therefore, collect part of the judgment from each of the physicians, or the entire amount from any one of them.
2. This is one reason why plaintiffs name as many defendants as possible. This is also why emergency physicians should avoid writing admitting orders; doing so only serves to extend their patient care responsibilities beyond the emergency department and increase their liability.

E. Statute of limitations

1. For every crime, except murder, there is a statute of limitations (SOL) that acts to limit the time in which an action can be brought. The SOL for torts such as negligence ranges from 1 to 3 years. Once the SOL has ended, the plaintiff is barred from bringing forth this cause of action.
2. The specific expiration date for an SOL depends on when it begins. Even though the SOL usually begins when the negligence or injury occurs, in some jurisdictions a discovery rule is followed.
 - a. Under the discovery rule, the SOL does not begin until a patient discovers (or should have discovered) injuries, even though discovery is delayed from the time of actual injury.
 - b. If a patient can show that fraud, misrepresentation, or deceit prevented discovery, an extension may be granted.
 - c. With minors, the SOL may not begin until the age of majority or emancipation is reached.

IV. MEDICAL RECORDS

- A. The Joint Commission defines basic standards of content for medical records. The records are considered legal documents that are admissible in court as evidence. Failure to maintain records as outlined below violates the standards imposed by Joint Commission accreditation. The following details represent the scope of information the physician is expected to provide the court.

1. Patient identification
2. Means and time of arrival
3. Vital signs and history of presenting complaint
4. Prehospital care
5. Therapeutic and diagnostic orders
6. Test and procedure results
7. Clinical observations (nonclinical remarks or emphasis markings should be avoided)
8. Treatment results
9. Diagnosis
10. Disposition and instructions
11. Documentation of facts concerning refusal of treatment

B. Although beyond the requirement, other items that should be recorded include:

1. Allergies
2. Medications
3. Last menstrual period
4. Tetanus immunization status
5. Name of primary care physician

C. Altering medical records

1. Mistakes can be corrected only while the patient is still under the physician's care in the emergency department by marking through the statement(s) with a single line, noting the reason for change (eg, "error" or "wrong patient") and initialing, dating, and timing the correction.
2. Nothing should be changed on a medical record after it is filed, even to correct mistakes. Supplementary records may be added for this purpose, but you should not change the original medical record. Changing or destroying medical records may constitute grounds for punitive damages.

D. Previous records

1. Patients have a reasonable right to rely on the availability of prior records, including diagnostic studies, when they return for treatment.
2. Failure to access previous records or studies may constitute breach of duty to the patient. (Example: Patient with a history of migraines dies of a subarachnoid hemorrhage hours after the condition was diagnosed as "migraine headache." Previous records demonstrate that the patient's headaches had never lasted 3 days.)

V. RESUSCITATION ISSUES

A. Initiation of CPR

1. In general, CPR should be undertaken whenever there is a possibility that the brain is viable.
2. The decision to withhold CPR should never be made in the field (even by a physician at the request of a family member) whenever there is a possibility that the brain is viable, unless a valid do-not-resuscitate order is available.

- a. A family member's accession of the emergency medical system manifests sufficient ambivalence if he or she subsequently asks that CPR be withheld in the ambulance.
 - b. The possibility of foul play must be considered when emergency medical personnel are asked to withhold CPR.
 - c. Hospital-based support mechanisms may enhance survival.
 3. In particular, the following patients are not necessarily dead and should be resuscitated:
 - a. Cold-water immersion arrest victims
 - b. Most pediatric arrests
 - c. Patients found in a cold environment with rigor mortis
 4. CPR can, however, be withheld and field pronouncement made when any of the following are present:
 - a. Decapitation
 - b. Decomposition
 - c. Dependent lividity
 - d. Incineration
 - e. Rigor mortis in a warm environment
 - f. Torso transection
 5. **Specific termination of resuscitation (TOR) protocols can be developed for prehospital providers and are used around the world.**
 - a. **Most agree that for pulseless adult patients without a shockable rhythm who have had an adequate trial of resuscitation, resuscitation may be terminated in the field.**
 - b. **Policies differ on the length of resuscitation attempts (generally 15–20 minutes) and the nature of the resuscitation depending on the level of the providers (EMT versus paramedics).**
 - c. **A policy for field TOR must include psychosocial support for the family and friends present.**
 - d. **Field TOR cases should be reviewed by a quality assurance process.**
- B. Reasons to withhold CPR in the emergency department**
1. A documented terminal illness for which all therapeutic options have been exhausted, in which CPR has been discussed by the patient or his or her family with the physician of record, and the decision has been made not to proceed
 2. A legally sufficient advance directive or durable power of attorney for health care supports the emergency department physician in withholding or terminating CPR.
- C. The decision to terminate CPR in the emergency department must be based on cardiovascular unresponsiveness as determined by the physician in direct contact with the patient. Correction of the underlying hypovolemia or other underlying complication should precede determination of unresponsiveness.**
- D. Pronouncing a patient**
1. Any licensed physician can pronounce a patient dead.
 2. Although the physician evaluating the DOA or the resuscitation team leader generally pronounces the patient, the patient's primary care physician can also be called on to do this; there is no statutory obligation for the emergency physician to pronounce the patient.

VI. MISCELLANEOUS ISSUES

A. Advance directives

1. The patient self-determination act of 1990 requires hospitals, upon patient admission, to inquire about the patient's advance directives, if any, and supply forms and materials for these if the patient so desires.
2. The three types of advance directives that the emergency physician is likely to encounter are living wills, do-not-resuscitate orders, and durable powers of attorney. Although some state-to-state variability exists, advance directives support the emergency physician in withholding life-sustaining support or treatment with minimal risk of legal consequences.

a. Living wills

- (1) Allow an individual of sound mind to control decisions relating to withholding or withdrawing of death-delaying procedures when the individual has a terminal condition
- (2) Are limited to terminal conditions and withdrawal of procedures that would only postpone the moment of death; they do not allow the withdrawal of food and water if such withdrawal would result in death solely from dehydration or starvation.
- (3) Are not effective if:
 - (a) There is a durable power of attorney and the agent is available, or
 - (b) The patient is pregnant and the fetus could potentially develop to live birth if death-delaying procedures are used.
- (4) Supersede the desires of the family or guardian
- (5) May be revoked by the declarant (the patient) at any time
- (6) Are usually honored out-of-state to the extent that the living will does not violate in-state provisions
- (7) Do not take effect unless the patient is in a permanently unconscious state or has a terminal condition as determined by two physicians

b. Durable power of attorney (POA) for health care

- (1) Provides an agent with broad powers to make healthcare decisions on behalf of the patient (the principal)
- (2) Becomes effective if and when the patient lacks the capacity to make decisions for himself/herself; however, it may become effective at a different time, as designated by the patient. The patient may lack capacity at some times and have it at others, so each situation must be evaluated for capacity. (For example, a patient who is delirious with infection in the ICU needs POA to make decisions but then improves, goes to a general medical floor, and is able to make his or her own decisions.)
- (3) Allows the agent to direct the withdrawal of nutrition or hydration
- (4) Takes precedence over the living will or family desires
- (5) Is generally upheld out-of-state to the extent that the general power of attorney does not violate in-state provisions

c. Do-not-resuscitate orders/physician orders for life-sustaining treatment

- (1) These are state-dependent orders signed by a physician after a discussion with the patient or the patient's POA or guardian.

- (2) Most states recognize orders from other states.
- (3) May be of different types, for example in Ohio:
 - (a) **Do-not-resuscitate Comfort Care Arrest:** a directive signed by a physician that states you are to receive standard medical care until you experience cardiac or respiratory arrest
 - (b) **Do not resuscitate Comfort Care:** a directive signed by a physician that states you are to receive any care that eases pain and suffering but no resuscitative measures to save or sustain life

B. Collection of medical evidence

1. Sexual assault evaluation

- a. The emergency physician's responsibility is to provide appropriate medical care and assist in the collection of evidence, while assuring that the patient's privacy is respected.
- b. Performance of the examination (including the acquisition of specimens for laboratory evaluation, the provision of necessary treatment, the retention of articles of clothing for evidence, and the release of information to the proper authorities) requires the patient's written consent and should be witnessed by another healthcare provider.
- c. Procedures for handling sexual assault investigations vary from state-to-state and must be closely adhered to, particularly those regarding maintenance of the chain of custody for the materials required by the state. The emergency physician can facilitate this by having one nurse involved in the process from start to finish; if only one nurse handles the specimens and then turns them over to a police officer, it best serves the chain of custody.

2. Toxicologic determinations

- a. Toxicologic studies should be ordered whenever they are clinically indicated to facilitate patient management.
- b. The governing statutes regarding the acquisition of a "legal" blood alcohol or drug level (one that is requested, usually by a police officer, as evidence) vary from state to state. In some states, the emergency physician may transmit blood or urine drug/alcohol test results to the state police or local law enforcement agencies. In other states, the emergency physician must convey this information; in other jurisdictions, the emergency physician must neither draw nor convey without consent or a valid court order. Make sure you are aware of the governing statutes in your state.
- c. When toxicology studies are obtained to be used as evidence, special collection and chain of custody procedures apply (see sexual assault evaluation above), and strict confidentiality must be maintained.

3. Family violence is defined as intentional intimidation (physical or sexual abuse) or the battering of children, adults, or elders by a family member, intimate partner, or caretaker. It includes child abuse and neglect, domestic abuse, and elder abuse, all of which involve injury of one family member by another.

a. Child abuse

- (1) The single most important element in diagnosing child abuse is a history that is inconsistent with the apparent injury.
- (2) The mechanism of injury and an accurate description of the injuries should be documented, with photographs if possible.
- (3) Whenever traumatic child abuse is suspected, a skeletal survey (including skull, chest, spine, and extremity films) should be considered.

- (4) All 50 states have statutes requiring the reporting of child abuse to the authorities, and most require reporting when there is a suspicion that abuse or neglect has occurred.
 - (a) All of these statutes provide immunity from civil liability for physicians who report suspected child abuse, but the level of immunity varies from state-to-state.
 - (b) Most child abuse statutes make reporting mandatory, with civil actions in negligence if physicians fail to report suspicions of child abuse; therefore, it is much safer medically and legally to report any suspicion of child abuse or neglect than not to do so.
- b. Child neglect
 - (1) Occurs when a parent (or some other person responsible for a child's welfare) does the following:
 - (a) Withholds or denies nourishment or medically indicated treatment (including food or care) *or*
 - (b) Does not otherwise provide necessary support (including clothing or shelter) or legally required education
 - (2) Reporting is mandatory and is part of the child abuse statutes in all jurisdictions.
- c. Domestic violence (also known as spousal abuse, partner violence, or battering)
 - (1) Defined as victimization of a person with whom the abuser has or had an intimate relationship
 - (2) Clues to domestic violence
 - (a) A mechanism that does not fit the injury
 - (b) Delay in seeking care
 - (c) Frequent trauma
 - (d) Substance abuse
 - (e) A history of child abuse in the family
 - (f) Patient evasiveness
 - (g) Minimization of the injury
 - (h) Depression
 - (3) In contrast to child and elder abuse, most states do not have reporting requirements for domestic abuse or immunity provisions to protect physicians who report such suspicions.
- d. Elder maltreatment: abuse or neglect of a person ≥ 60 years old by a caregiver or another person in a relationship involving an expectation of trust
 - (1) Clues to elder violence include those listed for domestic violence plus the following:
 - (a) **Conflicting reports by patient and caregiver**
 - (b) **Scald and cigarette burns**
 - (c) **Multiple fractures**
 - (d) **Injuries in various stages of healing**
 - (e) **Patient's inability to give a clear description of how the injury occurred**
 - (f) **Caregivers with a history of substance abuse or mental illness**
 - (2) Reporting of elder abuse is required in most states (>40) but is only voluntary in a few. Hence, it is important to be familiar with your state's statutes.

- (3) Immunity provisions also vary; some states follow child abuse immunity statutes, while others grant immunity only if the report is done in "good faith."
- (4) Maltreatment may include physical abuse, sexual abuse or abusive sexual contact, psychological or emotional abuse (social isolation, damaging property, or other controlling behavior), neglect, abandonment, financial abuse, or exploitation.

C. Telephone advice/triage

- 1. Considered to be a medical decision
- 2. Legally speaking, emergency physicians should resist the basic inclination to be of service and refuse to give medical advice over the phone.
- 3. The only advice that is prudent is to suggest that the patient be brought to a physician for a complete and thorough evaluation.

PHYSICIAN-PATIENT INTERACTION

I. ATTITUDES OF THE EMERGENCY PHYSICIAN

- A. Demonstrate a willingness to treat all patients regardless of the problem or clinical presentation.
- B. Demonstrate an empathetic attitude toward patients with difficult or complicated problems.
- C. Create rapport.
 - 1. Establish eye contact with your head and the patient's at the same level.
 - 2. Address the patient by last name to convey respect.
 - 3. Listen intently to fully ascertain the patient's agenda and concerns.
 - 4. Provide information and obtain feedback to ascertain comprehension and acceptance of your diagnostic impression.
 - 5. Explore treatment alternatives with the patient.
 - 6. Give the patient some control over the situation; be flexible in responding to his or her needs.
 - 7. Remain calm, especially if the patient becomes apprehensive, upset, or hostile.
 - 8. Provide the patient with a prediction of wellness.
 - 9. Discuss disposition and instructions with the patient before discharge.
- D. Errors in medical inquiry and clinical decision making
 - 1. Over 1 million patients each year are injured by preventable medical error, and 100,000 people die.
 - 2. Contributing factors
 - a. Overcrowding
 - b. Brief patient encounters
 - c. Chaos of a busy emergency department
 - 3. Broad categories of practitioner errors
 - a. Affective errors: pertain to physician attitude, and communication and conduct
 - b. Psychomotor errors: related to procedural or technical skills
 - c. Cognitive errors are related to errors in:
 - (1) Medical inquiry data
 - (2) Clinical decision making
 - (3) Faulty use of rules
 - (4) Faulty synthesis using the hypothetico-deductive decisionmaking process
 - 4. Ways to minimize errors in clinical decision making
 - a. Avoid relying on a previous diagnosis.
 - b. Avoid someone else's thinking whether it is related to diagnostic or personal bias.
 - c. Check for critical past medical history and risk factors for serious disease or poor outcome.
 - d. Pay attention to vital signs and notes of nurses and prehospital personnel.

- e. Avoid premature closure if the diagnosis is not certain; arrange for appropriate follow-up, and give specific instructions in written form.
- f. Beware of high-risk times: patient sign-out (see and touch all), high-volume or high-acuity times, and times of personal fatigue.
- g. Beware of high-risk patients: hostile, violent, or abusive patients; patients with alcohol or drug abuse; psychiatric patients; and patients who elicit a negative visceral response.
- h. Beware of the return visit; beware of what was missed during the previous visit.
- i. Beware of high-risk diagnosis: MI, pulmonary embolus, subarachnoid hemorrhage, tendon and nerve injuries, retained foreign bodies, intracranial hemorrhage in intoxicated patients, vascular catastrophes in older patients, appendicitis, meningitis, ectopic pregnancy, and testicular torsion. Exclude the worst-case scenario or high-risk diagnoses first.
- j. Beware of the nonfit: when the presumptive diagnosis does not match the symptoms, signs, or diagnostic tests; be willing to reevaluate the situation.

II. TRAUMATIZED VICTIMS OF VIOLENCE (SEXUAL ASSAULT, ABUSE, ETC)

A. Characteristic behavior

- 1. Patient may lose his or her sense of security.
- 2. Patient may experience distrust, guilt, or an irrational fear.
- 3. Patient may later develop a post-traumatic stress disorder.

B. Responsibilities of the emergency department staff

- 1. Listen sympathetically. Remember that most patients want physicians to ask about abuse and will answer truthfully if asked.
- 2. Communicate with the patient's family and friends.
- 3. Visit the patient often while he or she is in the department.
- 4. Make the patient as comfortable as possible.
- 5. Be especially sensitive to sexual assault victims.
 - a. Deal with their fears.
 - b. Provide support throughout the police interview.
 - c. Refer to rape counseling, social worker, or a psychiatrist.
 - d. Respect the patient's need for confidentiality in spite of the nonprivate nature of emergency department procedures and settings. Use an examining room that is isolated from the mainstream of the department. Have the same nurse assist you in all patient interactions.
 - (1) Ask police to talk with the patient only in the examining room.
 - (2) Fill out all forms in the examining room.
 - (3) Ask the patient if she would like a family member or friend to be with her in the room.
 - (4) Treat the patient kindly and with consideration.

- (5) Do not engage in conversation about the patient or the circumstances of the case outside the examining room. (For example, if you are standing in the hall, do not say to a nurse, "I have to do the rape exam before I see the next patient.")
- (6) Remember that reporting requirements for traumatized victims vary from state to state.
- 6. Be suspicious of injury presentations.
 - a. The most common sites of injury in victims of domestic violence are the head, face, and neck areas, as well as those usually covered by clothing (chest, breast, abdomen). Pregnant women are especially vulnerable.
 - b. Inquire about strangulation. It is a frequent form of domestic violence injury that patients often do not report unless asked; at least half these patients appear entirely normal, and only 15% display classic findings.
- 7. Provide detailed documentation; it improves quality of care, enhances availability of legal remedies for the victim, and helps with appropriate referral.

III. FAMILIES OF THE CRITICALLY ILL OR INJURED AND FAMILIES OF PATIENTS WHO DIE

- A. Need meetings with the emergency or attending physician and nurse
- B. Additional support from the emergency department staff
 - 1. Offer the families an opportunity to be in the room as invasive procedures and resuscitation are being performed.
 - 2. If family members choose to enter the room, clear their presence with medical staff and make sure the patient is accompanied by a staff member who focuses on the family members in the room.
- C. Qualities valued by patients when you are delivering bad news
 - 1. Attitude of the news bearer
 - 2. Clarity of the message
 - 3. Privacy of the conversation
 - 4. Adequate knowledge of the situation to fully answer family questions
- D. Viewing the body facilitates the grief reaction.
- E. Emergency department personnel need to facilitate
 - 1. Necessary papers to sign
 - 2. Organ donation
 - 3. Funeral arrangements
 - 4. Designing a plan to ease the burdens of surviving family members
 - 5. Dealing with the steps of the grief process (denial, anger, bargaining, depression, and acceptance)

IV. THE HOSTILE, ANGRY, OR UNCOOPERATIVE PATIENT

A. Types of physically threatening or angry patients

1. Delirious or intoxicated patients (90% of physically threatening patients)
2. Sociopathic patients (and those with personality disorders) who have a history of violence or acting out
3. Grieving patients who become angry
4. Frightened patients who become angry
5. Demented patients, children, and patients with limited mental capacity or developmental disabilities

B. Script for dealing with anger

1. Acknowledge that the patient is angry.
2. Note that the patient's anger seems justified (from his or her viewpoint).
3. Point out that the anger is working against the patient's care and the results he or she wishes to obtain.
4. Give the patient something to do so that he or she can become part of the solution.

C. Treatment

1. Identify patients likely to be violent, ie, demanding, agitated behavior, unrealistic expectations, forceful speech, or threatening body language.
2. Reduce sensory provocation for patients, eg, keep the police out of their view.
3. Reassure patients that they will not be harmed.
4. Do not be judgmental about patients' feelings.
5. Develop a staff plan for subduing and restraining violent patients.
6. Search any suspicious patients who may be carrying a weapon.
7. Work with the emergency department and hospital security personnel in developing a protocol for handling disruptive patients.
8. Do not allow patients to harm themselves or others; voluntary restraint is preferred over involuntary restraint whenever possible.
 - a. Ask patients if they feel that they might harm themselves or someone else; if so, ask whom they might harm and secure the safety of the intended victim when possible.
 - b. Ask patients if they wish to be restrained, and if so, how they would like to be restrained. (This "script for voluntary restraint" works only for patients who are not disoriented, able to cooperate, and accept restraints voluntarily.)

V. THE SUBSTANCE ABUSE PATIENT

A. Characteristic behavior

1. Disrupts departmental routine
2. Gives an unreliable medical history and makes the physical examination difficult to perform
3. Overreacts to real or imagined negative attitudes of staff

B. Treatment

1. Establish rapport and encourage the patient to discuss his or her alcohol or drug dependency.
2. Assess the patient's readiness to change.
 - a. If the patient is unaware of having a problem, denies having a problem, or is not ready to change, express concern and offer information, support, and follow-up options.
 - b. If the patient is unsure about whether or not to change, explore the pros and cons of change. If the cons of change outweigh the pros, ask the patient to think about his or her drinking behavior and encourage him or her to keep a diary of alcohol use; if the pros of change outweigh the cons, invite the patient to attend an educational program on the effects of alcohol use.
 - c. If the patient expresses a desire to change and is willing and ready to do so, help him or her implement a plan of action by providing specific resource information and conveying hope.
 - d. If the patient has relapses in sobriety, provide encouragement by reassuring him or her that relapse is a significant part of successful change and is considered to be part of a recycling process that leads to new behaviors.
3. Inform the patient of treatment alternatives.
 - a. Hospital, county, or custodial inpatient and outpatient detoxification units
 - b. Halfway houses
 - c. Self-help groups (eg, Alcoholics Anonymous)
4. Provide the patient with a medical follow-up plan and warn him or her of the effects of alcohol and drugs when taking prescribed medications.

VI. PATIENT DISPOSITION**A. Hospital admission**

1. 10%–15% of emergency department patients are admitted.
2. The emergency physician should consult with the patient's personal physician (if available), or the physician on call should be contacted.
3. Communication between emergency and admitting physicians should be explicit, documented, and include the time that the initial physician contact was attempted and the time the actual response was received.
4. ACEP has endorsed the principle that emergency physicians should not write admitting orders, because they extend the emergency physician's responsibilities beyond treatment in the emergency department as follows:
 - a. Legally, they can be construed to infer continuing inpatient responsibility on the part of the emergency physician.
 - b. They may involve the emergency physician in the management of patients in medical units remote from the emergency department where liability coverage may not extend.
 - c. They increase the likelihood that the patient will not be seen by the admitting physician in a timely manner.
5. If the emergency physician feels compelled to write admitting orders out of courtesy or other factors, such orders should be time limited and provide a clear transfer of responsibility to the admitting physician.

6. Occasionally, there are times when there are no inpatient beds, and patients must be "boarded" in the emergency department.
 - a. When this occurs, patients should still receive required care.
 - b. Hospital policies should be developed to prevent boarding and improve admission processes.
 7. Policies of using inpatient hall beds may be developed, because their use has not been found to result in patient harm.
- B. Definitive treatment for the discharged patient may include specialty consult to provide appropriate treatment while he or she is still in the emergency department and to determine disposition on discharge and outpatient treatment.

C. Transfers

1. The Emergency Medical Treatment and Active Labor Act (EMTALA) makes all hospitals with a Medicare participation agreement subject to this law and requires that any unstable patient transfer be medically indicated and not initiated for financial reasons. A transfer is "medically indicated" if the reason for transfer is to obtain a higher level of medical care necessary to treat the patient's condition (a level of care that is not available at the transferring facility). (See also rules governing emergency departments, pages 1176–1178.) Specific guidelines for transfer include:
 - a. The patient's condition should be stable. If the patient is unstable, then stabilization measures should generally be undertaken before transfer. If, in spite of these efforts, the patient remains unstable, he or she may be transferred only if definitive measures are unavailable at the transferring hospital, ie, the medical benefits outweigh the increased risks to the patient from the transfer.
 - b. The patient must be examined by a provider that the hospital's governing body has determined to be appropriate; this is often, but not always, a physician.
 - c. The patient must consent to the transfer unless he or she is mentally incompetent to do so.
 - d. Pregnant woman with contractions who have an emergency medical condition (as defined by EMTALA) may not be transferred until the baby and placenta are delivered, or the physician makes the determination that the benefit of transfer outweighs the risk of transfer of the mother or unborn child.
 - e. Communications between hospital (and physicians) should be fully documented and include the following:
 - (1) Facts establishing the need for transfer and the acceptance by the receiving hospital and physician
 - (2) Plans and provisions for protection and care of the patient en route
 - (3) Date and time of transfer
 - (4) Completed written transfer certification—this legal document must include items 1–3 above and an explanation of the risks and benefits of transfer.
 - f. Medical records must always accompany the patient (or follow shortly by electronic means if the transfer should not be delayed to gather the records).
 - g. The transferring physician is responsible for the patient's wellbeing until he or she arrives at (and is accepted by) the physician at the receiving hospital under the concept of abandonment. Under EMTALA, a hospital may also be held liable.

D. Ambulance diversion

1. A hospital or system may lack capacity to handle the current patient volume or type; thus, ambulance diversion policies may be created.
2. An agreement with other local hospital and prehospital providers
3. Per the ACEP policy on ambulance diversion, a hospital plan must include provisions for:
 - a. Situations in which hospital resources are not available and temporary ambulance diversion is required (loss of electricity or water, no inpatient beds available for admission, no operating rooms available for trauma, etc)
 - b. Notification of Emergency Medical Services (EMS)
 - c. Care for patients who continue to enter the EMS system
 - d. Notification of going off diversion
 - e. Consideration of solutions that address causes for diversion
 - f. Review of policies that govern diversion

E. Disposition problems based on nonmedical factors

1. Mentally or physically impaired patients
2. Patients without resources
3. Older patients who are unable to cope with their social environment (hospital-based social workers can best coordinate discharge plans for these patients)
4. Social admissions to ensure victim safety from the batterer when no shelter beds are available (Competency must be established before being discharged home.)

F. Other licensed supervised facilities

1. Skilled-nursing facilities
 - a. Offer IM or IV injections
 - b. Offer controlled oral medications
 - c. Can provide oxygen
 - d. Provide very close nursing supervision
2. Extended-care facilities (includes basic and intermediate nursing homes)
 - a. An adjunct to public facilities
 - b. Cost less than hospital care
 - c. Transfer between facilities should be made on a physician-to-physician level
3. Facilities that provide housing and meals
 - a. Homes for the aged (designed for healthy ambulatory seniors)
 - b. Adult foster care homes
 - c. Room and board homes
4. Emergency shelters
 - a. Located in metropolitan centers
 - b. Provide temporary shelter and sustenance for individuals and families
5. Services that provide in-home assistance
 - a. Visiting nurse association or public health agencies
 - b. Homemaker service
 - c. Meals on Wheels
 - d. Daycare center for seniors

6. Additional outpatient services
 - a. Geriatric psychiatric screening and counseling
 - b. Geriatric police escort service
 - c. Older adult transportation systems
 - d. Home health aide
7. The emergency department staff should assist patients with equipment and transportation problems.

PROFESSIONALISM

I. ADVOCACY

- A. Patient: There are multiple avenues for patients to seek advocates or for someone to provide a patient with an advocate.
1. Be familiar with local or hospital resources.
 - a. Hospital or extended-care facility ombudsman or patient advocate helps patients and families with complaints or concerns get answers or resources.
 - b. Social workers may help with placement and assistance with available state and local resources such as food, housing, and transportation.
 - c. Financial counselors may be available within hospital organizations to assist with payment options or signing up for state, federal, or local assistance programs.
 - d. Pharmacy may be available to assist patients with medication or allergy review and to review for interactions or inappropriate home medications.
 - e. Be aware of other local and hospital resources for specific patient populations.
 - (1) Lactation consultant/La Leche League
 - (2) Victims assistance
 - (3) Rape crisis
 - (4) Homeless shelters
 - (5) Safe houses
 - (6) Sober houses
 - (7) Battered women and children shelters
 - (8) Drug and alcohol assistance programs
 - (9) Developmental Disabilities Board
 - (10) Area Agency on Aging
 - (11) Children's services
 - (12) Geriatric consultants
 - (13) Smoking cessation programs
 - (14) Weight loss programs
 - (15) Dental clinics
 - (16) Discount pharmacies/local medication assistance programs
 2. Be familiar with regional and national resources.
 - a. National Patient Advocate Foundation: national nonprofit organization dedicated to improving access to healthcare through regulatory and legislative reform
 - b. Patient Advocate Foundation: national nonprofit organization that provides professional case management for individuals with issues of healthcare access, medical debt, or job retention related to illness
- B. Physician
1. Resources are available at local, state, and national levels.
 2. Many resources are available through medical societies such as ACEP, the American Medical Society, the American Medical Women's Association, etc.

- a. Legislative advocacy
- b. Regulation advocacy
- c. Expert witness advice
- d. Mentorship
- e. Leadership opportunities and training
- f. Continuing education
- g. Contract management/negotiation
- h. Work/life balance advice
- i. Policy statements
- j. Codes of ethics
- 3. State resources are available for physician impairment (see also well-being, page 1212).
 - a. State medical board
 - b. State medical societies

II. DIVERSITY AWARENESS

A. Cultural differences

- 1. Seek to understand different culturally beliefs and practices, while maintaining patient autonomy and legal obligations
- 2. Patients and colleagues should be treated with respect, regardless of color, gender, age, race, creed, religion, disability, sexual orientation, nationality, body habitus, or disease.

B. Conflicts

- 1. Occasionally, moral and ethical conflicts arise, such as a parent not wanting his or her child to receive a vaccine or treatment.
- 2. These situations must be dealt with according to hospital policy, local and state laws, and hospital ethics boards.

III. ETHICAL PRINCIPLES (BASED ON THE ACEP CODE OF ETHICS)

A. Morals

- 1. Set of guides that helps one make decisions
- 2. Professional oaths and codes of ethics may help one to decide the correct path.
- 3. Unique to emergency medicine are a lack of established patient-physician relationship, situations in which patients lack decision-making capacity, situations in which patient problems exceed available resources, acting as a health safety net for many patients, societal duty to render emergency aid outside of work, and by virtue of training and knowledge needed to perform emergency medicine, a resource for the community.

- B. Virtues: attributes of successful emergency physicians include courage, justice, vigilance, trustworthiness, and resilience.

C. Emergency medicine relationships

1. Patient duties
 - a. Beneficence (serve the best interest of the patient)
 - b. Nonmaleficence (minimize risk of harm)
 - c. Respect for patient autonomy
 - d. Justice (allocation of resources to maximize benefits and minimize burdens for all patients regardless of race, color, creed, or gender)
2. Other professionals
 - a. Duties of honesty, respect, and justice (for patients)
 - b. Oversight is also needed for physician extenders, prehospital providers, and anyone else involved directly or indirectly in patient care (nurses, administrative staff, registration, dietary, custodial staff).
3. Students and trainees: physicians should serve as teachers and role models for ethical behavior.
4. Researchers: research should be conducted in an ethical manner with oversight by Institutional Review Boards that protect the rights and welfare of patients and animals.
5. Society
 - a. Emergency medicine physicians have a duty to society to allocate resources fairly, oppose violence, and promote public health. These duties may sometimes come at an expense to individual liberties (eg, duty to report a communicable disease).
 - b. Local laws and policies may help guide decisions.
 - c. In the United States, the federal EMTALA law makes access to emergency medical care a fundamental right.
 - d. Outside the healthcare setting, Good Samaritan statutes protect healthcare professionals for good-faith efforts to render first aid.

D. Conflicts of interest

1. Physicians may have personal, financial, business, or professional interests that can interfere with their duties.
2. Interests should never affect patient care.
3. When engaging in teaching, public speaking, writing, giving opinions, and conducting research, all actual or perceived conflicts of interest, including those of immediate family members, should be clearly stated.

E. Medical errors

1. **Emergency medicine physicians should inform patients about errors and the consequences of those errors.**
2. **Should be done in accordance with hospital policies**

F. Expert witness

1. ACEP has an expert witness reaffirmation that upholds moral and ethical principles.
2. It includes being truthful and impartial, giving only testimony on matters that one is truly an expert on, evaluating care by generally accepted clinical standards, making every effort to determine a causal relationship between substandard care and outcome, submitting testimony to peer review if requested, and not receiving compensation based on outcome of litigation.

G. Gifts from industry

1. Gifts should be of minimal value and be of benefit to patients or educational function.
2. The integrity and judgment of the physician must be preserved; extravagant gifts compromise the physician's duty to serve the patient.

H. Impaired physicians

1. Mental or physical illness, including alcohol or chemical dependence, can interfere with a physician's ability to safely care for patients.
2. Hospitals, physician groups, and residencies should have written policies on the following:
 - a. Immediately removing an employee who is a safety risk
 - b. Reporting a suspected impairment
 - c. Investigating a suspected impairment
 - d. Returning to practice after a treatment
 - e. Promoting well-being and early intervention
3. Be aware of state licensing and credential requirements for treatment and recovery programs.
4. Physician-assistance programs are often available through state boards or medical societies to assist with investigation, treatment, and rehabilitation.

IV. ELECTRONIC COMMUNICATIONS AND SOCIAL MEDIA**A. Electronic communications guidelines**

1. All interactions with electronic communications and social media should respect patient privacy and the Health Insurance Portability and Accountability Act (HIPAA).
2. Electronic communications can be used to improve patient care but must be incorporated in a secure manner (eg, texting an image of a STEMI ECG to an interventional cardiologist without any patient identifiers).

B. Resources for guidance on social media for medical practice staff

1. State Medical Association
 - a. If a general narrative of a patient communication is posted on a social media site, it must be generic enough that the patient cannot be identified.
 - b. Be mindful of laws and regulations that apply to everyday work, such as the Civil Rights Act (prohibits discrimination based on race, color, sex, national origin, or religion), the Americans with Disabilities Act of 1990, HIPAA, and general tort principles (defamation, slander, libel, etc).
 - c. Limit "accepting" friends or followers: giving health advice on these sites might constitute creating an electronic record as a patient-physician relationship.
 - d. Do not assume that employer-owned computers, Internet access, or e-mail are private. Do not use company e-mail to send any material that may be seen as inappropriate for work.
 - e. Watch endorsements, and make sure your identity is clear to avoid violation of the Federal Trade Commission's Guides Concerning the Use of Testimonials and Endorsements.
 - f. Make sure you are familiar with your employer's and hospital system's social media and electronic communication policies.

2. Federation of State Medical Boards: policies to encourage physicians to protect themselves from unintended consequences and to maintain public trust
 - a. Protect the privacy and confidentiality of patients.
 - b. Avoid requests for online medical advice.
 - c. Act with professionalism.
 - d. Be forthcoming about employment, credentials, and conflicts of interest.
 - e. Be aware that information posted online (eg, pictures of a physician drinking alcohol) is available to anyone.

V. LEADERSHIP AND MANAGEMENT PRINCIPLES

- A. Many guides on how to be a good leader involve switching focus from "I" to "we" or "team."
- B. Leadership roles
 1. Traditional leadership roles focused on a top-down approach.
 2. Modern leadership roles focus on a collaborative approach with a partnership model. A leader seeks to empower and motivate others rather than demand performance.

VI. WELL-BEING

- A. Rights of emergency physicians: ACEP Policy
 1. Autonomy in clinical decision making shall be respected.
 2. Physicians have the right to expect adequate staffing and equipment.
 3. Physicians will be compensated for services and not be required to pay for privileges or referrals, nor shall they receive anything of value for referrals to others.
- B. Fatigue and impairment
 1. Fatigue can be an impairment (see also ethical principles, page 1209). Every physician should be mindful of his or her limits.
 2. Residents have work hour restrictions that teaching attending should be aware of.
 3. Schedules for emergency physicians should be created keeping in mind patient volume and the need for sleep between busy shifts. Strategies may vary by group (eg, 10 hours minimum between busy shifts, 24 hours off between 24-hour shifts, etc).
- C. Time management, life balance, and organizational skills
 1. Physicians are often busy and overstretched. Learning how to prioritize and organize can help with job and personal life satisfaction. Each person needs to decide which clinical, professional, and social activities are worth pursuing.
 2. Learning how to say "no" without feeling guilty may have to be learned and practiced. Seminars, lectures, and books are available and can help. A mentor or life coach might also be beneficial.

D. Work dysphoria (burnout)

1. In the United States, 30%–40% of physicians experience some degree of burnout.
2. Work-related stress causes burnout.
3. Increasing paperwork burden, decreased autonomy, decreased reimbursement, lack of tort reform, problems with work/life balance, and physician shortages may all contribute to burnout.
4. Medical schools, medical societies, hospitals, and residency programs should have policies to promote physician well-being and patient safety.

OTHER CORE COMPETENCIES OF THE PRACTICE OF EMERGENCY MEDICINE: PRACTICE CLINICAL SCENARIOS

Answers immediately follow the practice clinical scenarios.

Scenario A

Presentation: A patient presents to your hospital in labor, but her insurance does not cover admissions.

What law requires that your hospital handle the delivery of the fetus and placenta before she and the baby can be transferred?

Scenario B

Presentation: A patient is brought in by police for calling 911 to express suicidal ideation. The patient has ongoing suicidal thoughts and auditory hallucinations. The patient states he is going to kill his brother and gives his name. The patient requests discharge.

What should the physician do?

Scenario C

Presentation: An elderly patient presents from home via ambulance with a chief complaint of fever. Examination reveals infected bedsores, soiled clothes, and unkempt appearance. The patient tells you she lives with her son, who feeds her once a day and uses her social security check to buy his cigarettes. She is bedbound from a previous stroke.

What is the diagnosis?

ANSWERS TO PRACTICE CLINICAL SCENARIOS

Scenario A

EMTALA

Key facts: Under EMTALA, the patient and her infant can be transferred once medically stable. If the infant is unstable, all attempts to stabilize him or her must be made. If there is no inpatient neonatal unit, the infant can be transported even if he or she remains unstable if:

- You as the treating physician certify that the medical benefits outweigh the risks.
 - You send copies of the medical records.
 - The receiving hospital has the space and personnel to treat.
 - The receiving hospital has agreed to accept the transfer, and
 - The transfer will be performed by qualified personnel with appropriate equipment.
-

Scenario B

Mental health

Management: The physician has the duty to treat the patient and to warn the patient's brother. The physician cannot discharge the patient despite the patient's request because of the potential harm to the patient and the brother. The patient can be committed against his will for a fixed period of time. The physician should attempt to contact the brother and the police about the patient's homicidal thoughts.

Scenario C

Elder maltreatment

Management: You check your state laws and you have mandatory reporting of elder abuse. You contact the appropriate agency that will investigate. You admit the patient for treatment of her infected bedsores and initiate a social work consult.

NOTES

MECHANICS OF THE WRITTEN BOARD EXAM

General Information

The written board exam in emergency medicine tests your knowledge base relative to predetermined criteria of clinical competency. Consequently, there are two critical aspects of this test:

1. What is the written exam actually testing?
2. How are you being evaluated?

The written board exam measures six outcomes of learning and assesses five goals of learning. The outcomes of learning that can be assessed for any content area include:

- Knowledge
- Understanding
- Analysis
- Application
- Synthesis
- Evaluation

In addition, multiple-choice exams usually assess the goals of learning, which include:

- Knowledge of terminology or specific facts
- Knowledge and application of methods and procedures
- Ability to apply facts and principles in a given situation
- Ability to interpret cause and effect relationships

The written board exam tests both the outcomes of learning as well as the goals of learning. This text was designed and written with this in mind, which is why it is a teaching text (not just a reference text). In addition to providing facts, special attention has been given to the reader's need to understand concepts and to think critically, thus improving the ability to analyze, apply, synthesize, and interpret information. This is essential to effective learning, which is what this exam is really testing.

The second critical aspect of the written board exam is understanding how you are being evaluated. Some written exams discriminate among individuals, ranking them on relative levels of learning. These exams are "norm referenced" and are designed to discriminate in a way so that a certain percentage of candidates will not pass. The written board exam in emergency medicine does not discriminate; it is "criterion referenced," which means that there is no ranking, ie, 100% of candidates can pass. It describes what an individual can or cannot perform, with the difficulty of the test item matching the difficulty of the task.

The academic content of this exam is determined by a test development team composed primarily of physicians who practice emergency medicine. This team of emergency physicians has selected specific topics and ranked them in order of importance to the practice of emergency medicine. Over time, the test development team has modified these topics somewhat but not much. While the percentage of the types of exam questions has changed since the first exam (and on subsequent exams), the exam content has actually changed very little. In June 2001, the list of specific

topics was overhauled. This resulted in the development of the Model of the Clinical Practice of Emergency Medicine (EM Model), which was basically a reorganization of the topics and subtopics within a new frame that lists "Conditions and Components." This list describes the relative weight given to the topics of the EM Model in preparing the written board exam.

The content of the written board exam is based on a national (not regional) standard of care. Regardless of where you practice or the patient volume you encounter, you will be tested on a "national norm," which we have adhered to in this text.

Exam Format and Scoring

The Qualifying Examination is a comprehensive examination that covers the breadth of emergency medicine. The examination contains approximately 305 single-best answer multiple-choice questions. Between 10% and 15% of the questions will have an associated pictorial stimulus. The qualifying examination is administered at approximately 200 Pearson VUE professional computer-based testing centers throughout the United States and is administered once per year, usually in the fall. To accept a qualifying examination assignment, physicians must register online through the ABEM website using instructions sent with the assignment. Registration deadlines and fees are listed under Qualifying Exam Dates and Fees.

Once you complete registration, the ABEM online system will display the necessary information for scheduling an appointment. Approximately 30 days before the qualifying examination, ABEM will send each physician with a confirmed registration the Examination Information for Candidates book. It includes confirmation of examination seating, examination logistics, and other information about the examination. Each examination appointment is approximately 8 hours long with approximately 6 ½ hours devoted to testing. Images are presented along with the relevant test question on the computer screen.

To pass the exam, you need a score of 75% or higher. Remember that only one answer is correct, and it is presumed to be the single best answer. If you mark more than one answer, the question will be scored as incorrect. Consider the following:

1. After the test is given, an analysis of the results of testing is conducted so that poorly designed questions can be discarded before scoring.
2. There is no penalty for guessing; no points are subtracted for an incorrect answer.
3. Field testing of certain questions (for purposes of research and validation) is done every year; you are not likely to identify these questions (nor should you try), and they do not count toward your score.

All of this is in your favor. It means that you do not have to answer 75% of all the questions to get a "score" of 75%. If you answered all the questions and if you felt reasonably confident that you answered 60% or more questions correctly, chances are that you passed. For example, if there are 335 questions and 135 of them are field test questions and questions that have been thrown out, only 200 questions "count." In other words, a score of 75% is not 75% of 335 (251) but 75% of 200 (150).

The Continuous Certification (ConCert) Examination is similar to the former recertification examination. There are, however, important differences. It is also administered at approximately 200 Pearson VUE professional computer-based testing centers, but it is a shorter examination of approximately 205 multiple-choice questions.

The ConCert examination is a comprehensive examination that covers the breadth of emergency medicine. Each examination appointment is approximately 5 ¼ hours long, with 4 ¼ hours devoted to actual testing time. Between 10% and 15% of the questions will have a pictorial

stimulus. The style of the test questions is identical to that of the Qualifying examination test questions, ie, single-best answer, positively worded, multiple-choice questions focused on what the practicing emergency physician needs to know when treating patients.

The ConCert examination is a criterion-referenced examination. All candidates achieving a final score of 75% or greater pass the examination.

The link between previous LLSA readings and the ConCert examination no longer exists. ConCert examinations are focused on assessing knowledge needed for clinical practice. Although LLSA questions no longer appear on the ConCert examination, similar concepts may still be represented as they become the standards for practice in emergency medicine. The questions of detailed information found on the LLSA tests, however, are not found on the ConCert examination.

To see your individual ABEM MOC requirements and check your eligibility, go to www.abem.org, sign in using your user ID and password, select MOC Online, and view your requirements and status page.

LLSA Content

The primary goal of LLSA is to promote continuous learning. Readings are intended to address issues relevant to current clinical practice at the time they are posted. LLSA readings are designed as study tools and should be read critically. They are not intended to be all inclusive and are not meant to define the standard of care for the clinical practice of emergency medicine.

A list of 10 to 15 readings based on the EM Model is posted on the ABEM website each year. (Prior to 2010, reading lists consisted of 16 to 20 readings.) LLSA tests consisting of 20 to 30 questions are developed based on the annual readings. (Prior to 2009, LLSA tests consisted of 32 to 40 questions.)

Each LLSA reading list will be posted approximately 1 year in advance of its associated LLSA test. A new LLSA test will be posted on the ABEM website in April of each year. Each LLSA test will remain online for 3 years.

Beginning with the 2014 LLSA test, readings will no longer be organized by designated and non-designated content areas, but rather will be solicited from all areas of the EM Model. Prior to 2014, each year of the 9-year LLSA cycle focused on specific areas of the EM Model.

Registration for and taking of LLSA tests is done online through ABEM's website. Beginning with the 2008 test, a passing score is achieved by answering 85% of the items correctly. Once registered for an LLSA test, diplomates have three opportunities to pass it. If necessary, a diplomate may register and pay the test fee again for additional opportunities to pass a test. Each test will remain online for 3 years, based on the date of publication. When a test is retired, the associated reading list is also retired and is no longer available online.

Osteopathic Board Certification Process

Initial certification for graduates of osteopathic emergency medicine residency programs is offered through the American Osteopathic Board of Emergency Medicine (AOBEM), the certifying body of the American Osteopathic Association. The process involves a comprehensive written examination (Part 1), an oral examination (Part 2), and a clinical chart review (Part 3).

In support of lifelong learning, the AOBEM also requires a certification process similar to the LLSA known as Continuous Osteopathic Learning and Accreditation (COLA) examinations. A formal written recertification examination is also required every 10 years. Details regarding these procedures are available on the AOBEM website (www.AOBEM.org).

AOBEM offers the initial Certification Examination (Part 1) and the written Formal Recertification Examination at Prometric computer testing centers throughout the country. The more than 300 professional standardized test centers are located only within the United States (all 50 states).

The Certification Examination Part 1 is a computer-based test usually offered in mid to late March. The examination dates can be found on the AOBEM web site (www.AOBEM.org) under the Important Dates section. After successful completion of Part 1 of the written examination, candidates will be notified of the date and time of the oral examination.

The Formal Recertification Examination is a combined computer-based test and oral examination. It is offered once a year. AOBEM recommends that candidates register at least 90 days in advance of a scheduled test date, although candidates may register as early as 6 months in advance. The computer-based examination covers the breadth of the clinical practice of emergency medicine and consists of approximately 125 questions. The time limit to complete the test is 2 ½ hours. The oral examination includes four clinical cases. You must pass both the computer-based and oral portions of the examination to achieve recertification.

The AOBEM Table of Specificity serves as the basis for the content of both the written certification and recertification exams.

AOBEM Specificity for Certification/Recertification Written Examination*

	Core Content Categories	Ranking
1	Abdominal and Gastrointestinal Disorders	H
2	Cardiovascular Disorders	H
3	Cutaneous Disorders	L
4	Endocrine, Metabolic, and Nutritional Disorders	M
5	Environmental Disorders	M
6	Head, Ear, Eye, Nose, Throat Disorders	M
7	Hematologic Disorders/Immune System Disorders	L
8	Systemic Infectious Disorders	H
9	Musculoskeletal Disorders (Nontraumatic)	M
10	Nervous System Disorders	H
11	Obstetrics and Disorders of Pregnancy/Gynecology	M
12	Pediatric Disorders	H
13	Psychobehavioral Disorders	L
14	Renal Disorders/Urogenital Disorders	L
15	Thoracic and Respiratory Disorders	H
16	Toxicologic Disorders/Clinical Pharmacology	M
17	Traumatic Disorders	H
18	Administrative Aspects of Emergency Medicine/EMS	L
19	Disaster Medicine	L
20	Procedures and Skills	M

*Entire table can be found online at www.AOBEM.org.

Note: H, M, and L indicate topics considered to be of high, medium, and low importance, respectively.

The AOBEM Certification Examination Part 1 has 286 multiple-choice questions. You will have 4 hours to complete the morning session and 2 hours to complete the afternoon session.

The whole examination is divided into six sections, with the first five sections consisting of 50 questions and the last section consisting of 36 questions. Within a section you can answer questions, mark questions so you can go back to them later, skip questions, go back to review previously marked or answered questions, and change answers. At the end of a section, you will be asked to click the END SECTION button. Once you choose to end the section, your answers to the questions in the section will become permanent, and you will not be allowed to go back to that section. There is no time limit to complete any single section. Since you must finish the morning session (four sections) in 4 hours and the afternoon session (two sections) in 2 hours, the AOBEM suggests that you spend no more than 1 hour on each section.

You will have the option of taking a 10-minute break between Sections 2 and 3. If you choose to take the break, the actual time you spend on the break will be deducted from the time allocated to complete the examination. During the break, you must leave the testing room. When you reenter the testing room, you must show a valid ID and a fingerprint will be obtained.

Following the morning session (after Section 4), there will be an optional 40-minute lunch break, before the afternoon session begins. If you spend less than 40 minutes on the break, the actual time you spend on the break will not be deducted from the exam time. If you spend more than 40 minutes on the break, the afternoon exam session will begin after 40 minutes has passed. During the break, you must leave the testing room. When you reenter the testing room, you must show a valid ID and a fingerprint will be obtained. You will be responsible for managing your own time. If you take a break, please allow sufficient time to check in and be seated.

The Formal Recertification Examination is divided into three sections. The first two sections consist of 50 questions, and the last section consists of approximately 25 questions, but this can vary. Within a section you can answer questions, mark questions so you can review them later, skip questions, return to review previously marked or answered questions, and change answers. At the end of a section, you will be asked to click the "End Section" button. Once you end the section, your answers to the questions in the section will become permanent and you will not be permitted to return to that section. There is no time limit to complete any single section. However, because you must finish all three sections (the entire examination) in 2 ½ hours, AOBEM suggests spending no more than 1 hour on the first two sections and no more than 30 minutes on the third section. Questions in each section are grouped by question type, with independent one-best-answer questions first, followed by one-best-answer question sets.

Both the certification Part 1 and recertification questions are multiple-choice questions. Every question includes a set of choices, including one correct answer and several distracters. Most examination questions consist of five choices, although a few may have four choices or more than five choices. Certification Part 1 questions may be presented in a stand-alone format and an item set format. The recertification questions are all stand-alone questions. These items consist of a stem, a lead-in question, and several choices, one of which is the correct answer to the lead-in question.

AOBEM has established a predetermined pass rate; examination scores are not curved in any way depending on candidate performance on any examination administration.

Candidates will receive an official copy of their score reports from the AOBEM by mail within 4–6 weeks of their examination date. The candidate score report will provide a 3-digit total score, and a pass/fail designation. Passing the AOBEM Certification Examination Part 1 is based on an examinee's performance on the total exam, not on the performance of each topic. However,

candidates will be provided with a graphic performance profile according to the AOBEM content categories. The performance profile provides information on an examinee's performance on each topic compared with others who take the same exam. The examinee may use the profile to assess areas of strengths and weaknesses.

Certification Through the American Board of Physician Specialties

The American Board of Physician Specialties (ABPS), the official certifying body of the American Association of Physician Specialists, Inc. (AAPS), offers computer-based (written) certification examinations for the Board of Certification in Emergency Medicine (BCEM) twice a year and oral examinations once a year.

The examination process in emergency medicine requires candidates to pass both a computer-based (written) examination and an oral examination. The computer-based emergency medicine examination is offered twice each year at a network of testing centers throughout the United States and Canada. The exams are offered in May and November, during month-long testing windows. ABPS sends written examination results to candidates generally within 30–45 days of the test date, with earlier reporting options available for unofficial results. Candidates must successfully complete the written exam before being approved to sit for the oral exam. ABPS administers BCEM oral examinations in Tampa, Florida, once a year in early April.

The written examinations begin as the candidate schedules, provided the testing center is open for the appropriate amount of hours. The written exam is administered in two 4-hour sessions, with a break between the sessions, but the two sessions must be taken in the same day. Candidates are advised to schedule the exams as early in the day as possible.

The written examination consists of approximately 350 multiple-choice questions of four choices. (Experimental or nonscorable items may also appear on an examination. These questions will not necessarily be identified as nonscorable.) Each scorable question has only one correct answer. For each session, candidates are provided online access to approximately half the questions. A tutorial is provided to help the candidate understand the online process. ABPS also provides a formal comment form on which a candidate can enter comments regarding any question. All comments are read to determine if a question may be flawed. Upon review of the comments and item analysis (statistical data), if an item is considered flawed, it is removed from the scoring of the examination. The passing score on forms of the examinations varies depending on the expected performance values of the individual questions on the examination. Currently the minimum score for passing, depending on the form of the examination, ranges from approximately 75% to 82%, with each response to a question being scored correct or not correct, with no penalty for guessing in the calculation of the final score. Candidates receive score reports indicating their pass/fail status on the examination. Candidates who fail receive a breakdown of their performance by subject content domains and which performance on each domain met an expected minimum level of performance.

The following table lists the approximate number of items in each domain included on each form of the written examination. Cases for the oral exam are also drawn from these domains and use the same reference materials.

	Domain	Number of Questions
1	Administrative and Legal Aspects, Disaster Medicine, and Emergency Medical Services	9
2	Cardiovascular Disorders	38
3	Dermatologic Disorders	8
4	Ear, Nose, and Throat Disorders	18
5	Endocrine, Metabolic, and Nutritional Disorders	8
6	Gastrointestinal and Abdominal Disorders	24
7	Hematologic, Oncologic, and Immunologic Disorders	7
8	Infectious Diseases	13
9	Nephrologic Disorders	11
10	Neurologic Disorders	21
11	Obstetrics and Gynecology	21
12	Ophthalmologic Disorders	10
13	Orthopedic Disorders	21
14	Pediatric Disorders	18
15	Pharmacology	8
16	Procedures and Skills	8
17	Pulmonary and Respiratory Disorders	21
18	Psychiatric and Behavioral Disorders	10
19	Toxicology and Environmental Disorders	22
20	Traumatic Disorders	44
21	Urogenital Disorders	10

How To Take a Multiple-Choice Exam

Your skill in taking a multiple-choice exam is as important as adequate preparation. A common misconception is that all candidates who fail this type of exam do so because they have failed to master the subject matter. In truth, some test-takers have not learned how to apply their knowledge to a multiple-choice exam. For those of you who have previously encountered some difficulty with this type of exam, included are some general recommendations as well as specific test-taking strategies.

Remember

1. The exam is testing basic knowledge, not sophisticated, new age issues. Know the basics.
2. This is an exam for emergency physicians.
3. Absolute contraindications are more important to know than relative contraindications.
4. You generally need to read things five times to store them into long-term memory.
5. Associations are important on board exams. Learn them! You cannot ask for any additional information when taking the exam—no new laboratory studies, radiographs, etc. The exam question writer has given you things that you can associate with whatever condition the question is asking about. Here are some examples of associations:

- a. *Pseudomonas* osteomyelitis is associated with a puncture wound through a running shoe but not with a simple soft-tissue infection.
- b. Diarrhea caused by *Campylobacter* is associated with Guillain-Barré syndrome.
- c. Humeral shaft fracture is associated with wrist drop due to radial nerve injury.
- d. Ring-enhancing lesions on CT brain are associated with toxoplasmosis, neurocysticercosis, and the correct choice.

These are just a few examples of many. But be assured that ABEM loves associations.

- 6. Spend your time on the important topics. The list below represents almost 50% of the total exam. Look at the ABEM website for the rest.
 - a. Trauma accounts for 11% of the exam
 - b. Cardiovascular for 10%
 - c. Signs and symptoms for 9%
 - d. Abdominal/GI for 9%
 - e. Thoracic and respiratory for 8%
- 7. Spend your time on what is important.
 - a. Most common
 - b. Triads, pentads
 - c. Deadly causes
 - d. Complications
 - e. Associations
 - f. Worst-case scenario for a given presentation
 - g. Things we do that may kill
 - h. Things we do not do that may kill
 - i. Things we get sued for
 - j. ACLS, ATLS, PALS

General Recommendations

- 1. Learn how to relax while taking the written exam. If you feel tense before the exam actually begins, take a few seconds to induce a sense of positive self-expectancy. Say to yourself, "I will do well because I expect to do well." If you expect to do well, the odds are that you will.
After you have induced a sense of positive self-expectancy, close your eyes, take a deep breath, and tell yourself to relax. As you begin taking the exam, some of the tension will dissipate spontaneously simply because the conscious mind cannot do two things at once; you cannot think about being tense while you are answering questions. To stay relaxed during the exam, work rapidly but carefully. If you are not sure of an answer, skip that one and come back to it later. This is very important psychologically. Answer all the questions you know first and then go back later to work on those you don't know.
- 2. Avoid fatigue. Have a good night's sleep before your exam day. Because the exam is a long one, give yourself a rest period every hour or so, even if it is a very short one. Relax in your seat, and close your eyes for a minute or two. These rest periods will help maintain your mental efficiency at a high level.

Specific Test-Taking Strategies

If you do not know the answer to a question or if you are not sure of the answer, move on to the next question. When you return to the question, guess. Although guessing will probably not

significantly improve your score, it will not lower it. There are guidelines that can help you select the best answer so that if you are unsure of the answer, you may guess smart (see below).

The first thing you have to determine is if you understand the question but don't know the answer, or if you are not sure what the question is asking.

Let's begin with questions you understand but are unsure of the correct (best) answer:

1. Eliminate obviously incorrect answers. Then concentrate on the remaining answers.
 - a. An answer that "doesn't fit" with the other answers is probably wrong.
 - b. A wordy answer that is unclear or does not read well is probably wrong.
 - c. An answer with the words "always" or "never" is probably wrong (but these are rarely found in tests today because they were recognized as "give aways").
 - d. An answer that does not correlate grammatically with the question is probably wrong.
2. If you have eliminated obvious and probable incorrect answers and you still have more than one answer remaining, try looking at the question and completing it mentally before looking at the answers.
 - a. A spontaneous completion that corresponds to one of the answers is usually correct.
 - b. A spontaneous completion that is similar to two answers suggests that one of them is correct. Determine which one best completes the statement.
 - c. A spontaneous completion that is opposite to one of the remaining answers eliminates that choice.
3. Look at adjectives and adverbs in the question. Descriptive (key) words are frequently clues to the correct answer. Words like "acute" or "classic" or "routinely" are examples. This is a particularly valuable strategy if you are dealing with a question that seems to have two possible answers (the key word will fit one answer better than the other). Another example is "definitive" treatment versus "initial" treatment.
4. If you encounter a question containing a clinical presentation and you are asked to make a diagnosis based on an image (ECG, radiograph, photo of a skin lesion, etc) but you are not sure what the image shows, try to answer the question without looking at the picture. Then confirm the answer by looking at the picture. The correct answer should "match" the clinical presentation.
5. If the question covers a subject you know little or nothing about, see if there are other questions within the examination that include the same material. You can sometimes find useful information contained in other questions that can help you.

Points to Consider About Answers

1. If there are numbers contained in the answers and two answers have similar numbers, one of them is usually the correct one.
2. If one of the answers is "all of the above," look for two correct answers. If you find them, "all of the above" is the correct answer.
3. If one of the answers is "none of the above," look for one correct answer. This excludes "none of the above," and the one correct answer is the answer that is wanted.

All of these techniques can be used to answer questions you understand. Now let's deal with questions you don't understand.

Some questions are designed to test your ability to think critically or analytically. The answers to these questions are not obvious—they are inferred. Here's an example:

"What is the best pharmacologic choice for the patient with supraventricular tachycardia and hypertension?"

- (a) Adenosine
- (b) Digoxin
- (c) Methoxamine
- (d) Edrophonium

If you look in standard emergency medicine textbooks, you will not find a drug listed for the treatment of supraventricular tachycardia with hypertension. You will find, however, several drugs listed that should not be given to patients with hypertension: all the vasopressors (methoxamine, norepinephrine, metaraminol, phenylephrine, etc), digitalis preparations, and edrophonium chloride. Therefore, the question does not ask you to directly identify the treatment of choice. Instead, the question asks you to infer the treatment of choice by eliminating the agents you should not use; the agent remaining will be the correct answer (adenosine).

Here is another example of an "inferred" answer:

"What drug should be used in the patient with CHF who also has signs of digitalis toxicity?"

- (a) Furosemide (Remember, the question is about digitalis toxicity, not CHF.)
- (b) Quinidine
- (c) Nitroprusside
- (d) Propranolol

Again, there is no "drug of choice" for patients with CHF who are digitalis toxic. There are, however, drugs that should not be used in the presence of digitalis toxicity. Quinidine and calcium channel blockers (nifedipine, verapamil, and diltiazem) decrease digitalis clearance, so they should not be used. Furosemide tends to induce hypokalemia, which increases the risk of digitalis toxicity, so this is not a good choice. β -blockers (propranolol, nadolol, metoprolol, atenolol, timolol) are contraindicated in patients with severe CHF and should not be used. On the other hand, vasodilators (nitroglycerin, nitroprusside, prazosin, and hydralazine) lower systemic vascular resistance and improve cardiac output. The correct answer, therefore, is nitroprusside.

In developing any skill, practice is important. Practice answering multiple-choice questions on a regular basis. It is not only a good way to assess your knowledge base, but it is also a training mechanism for developing test-taking skills. As you go through the current *PEER* products, apply the techniques you have learned here when confronted with problem questions. With practice, this skill will improve, and you will feel more confident about your ability to use it effectively.

On any given exam, test-taking strategies can be successfully applied to 5%–15% of the questions. Some candidates fail this exam by only one or two points (score of 73 or 74). This amounts to about 15 to 30 questions. Because there are 310 to 340 questions on the exam, about 30 to 40 questions could be answered using these techniques, which might be just enough to help you pass.

ADDITIONAL TIPS FOR GOOD PERFORMANCE

Preparing for the written board exam is an arduous task, one that requires intense review and study. In some respects, the final phase of preparation is more difficult than the earlier ones. For one thing, you are tired. In addition to studying, most of you are working full time. Another problem is that your anxiety level is likely to peak as the exam date draws near. Finally, your self-study plan/preparation may leave you with a feeling of being overwhelmed by the amount of academic information you have been learning and relearning.

In this section are some guidelines to follow in the time remaining before the exam. It is recommended that you map out a program that will reduce your fatigue, lessen your anxiety, and allow you to focus your attention on the most important academic information you need to know.

Self-Care

Good health habits are important. If you have unhealthy habits, it will become increasingly harder to study and you will retain less.

1. Get plenty of sleep.
2. Eat more fiber-containing foods and less sugar and fat. You are more likely to be alert when you study as well as have greater endurance.
3. Engage in some form of mild exercise. Walking is excellent if you are not a runner; aerobics and swimming are also helpful.

Positive Self-Expectancy

A positive attitude is also important. Your attitude, both before and during the exam, can affect your exam score. A negative attitude will foster procrastination before the exam and will interfere with your performance during the exam. On the other hand, a positive attitude will facilitate your preparatory efforts and enhance your performance during the exam.

A technique that can help you build and maintain a positive attitude for the written board exam is called "mental rehearsal," and it can be very effective in reaching a goal and achieving success.

Mental rehearsal consists of previewing an upcoming event in a positive frame of mind. This should be done during the weeks before the exam. The purpose is twofold: to allow you to prepare for the exam as completely and thoroughly as you can in the allotted time, and to maximize the probability of a good performance on the exam day.

The art of positive self-expectancy is one that is practiced by many professionals before a major event. Essentially it consists of mentally rehearsing a perfect performance during the days, and even weeks, before the actual event. To use this technique, set aside time each day when you are completely alone and relaxed and imagine yourself taking the exam in a calm, confident manner with the expectation of doing well.

When you use the mental rehearsal technique, concentrate on what you know rather than on what you don't know. Do not worry about subjects or areas that you didn't have time to study. Rather, focus on what you have studied and then make some affirmations about your ability to do well on exam day: "I will do well on the exam. I have studied and worked hard to get where I am today and now, I'm going to go further still. I can do it." Positive affirmations have a powerful effect on the subconscious mind because they foster our belief in ourselves, which translates into confidence in our ability.

Don't put yourself into a position to be discouraged when you start rehearsing. Just do your best. Within a short time, the result of this technique will speak for itself, providing you with all the justification you need.

Written Board Courses

Many state ACEP chapters (and other organizations) conduct courses for emergency physicians taking the written board exam. These are intensive programs (4–7 days) that provide a comprehensive review of emergency medicine. They are usually given about a month before the exam. The utility of these courses varies from one course to another and from one physician to another. Not everyone needs or benefits from a written board review course. Many emergency physicians have passed the exam without it.

The Ohio Chapter ACEP course has had a reputation for over 30 years as one of the best reviews in emergency medicine (*EM Resident*, Emergency Medicine Residents Association, August/September 2012). Ohio ACEP offers a 5-day comprehensive academic review of emergency medicine, as well as a 3-day course that focuses on key facts and pearls of each emergency medicine topic.

The ACEP master calendar (www.acep.org) can provide information on a course that suits your schedule and location needs. Also, EMRA puts out a periodic review of board review courses; refer to them as a reliable, independent resource to select a course.

How to Use an Emergency Medicine Written Board Course

If you attend a written board course without any prior preparation, the program can be overwhelming. A lot of material is delivered very fast. If you have been reading this text before the course, your task will be a lot easier.

Read as much of this text as possible before the course. Remember to highlight key concepts and important facts. Take this book with you to the course; this is your base on which to build. Use the book as your guide during the course.

If you are attending an ACEP course, take the daily pretests and grade yourself; participate in evening review sessions, which cover hundreds of board-type questions. If you missed any questions, determine whether you didn't know the material or you did know the material but misread (misinterpreted) the question. Pay attention to pretest scores that are lower than 75% or several questions you did not know on certain topics. These are weak areas.

For those of you who have been studying for more than 3 months, listen to your speakers very selectively. Listen for information that is new to you. If you have been studying with some regularity the last few months or so, your database is fairly strong. The course will not only be a review for you but will also fill gaps in your knowledge base. Use this text to write in new information that seems important.

For those of you who have been studying less than 3 months, a different approach may be more helpful. If at all possible, review the syllabus material before each lecture. While you are listening to the lecture, take notes and mark your syllabus (underline) important information. Later, insert the syllabus material in this text.

For all of you: From the time you finish the course until a couple of days before the exam, review only the highlights of the study materials you have been using; it should all be in this text now.

1. Review any material that is likely to be on the exam, ie, any information that your speakers pointed out as "must know for the exam." You don't want to miss questions that you expect to see on the exam.

2. Review any information you learned in this text or during the course that clarifies your understanding of a particular subject. This is important because if you encounter questions on this topic and you are not sure of the answer, you will be able to figure it out.
3. Review any area where you are weak. Do not pick more than three because you do not want to "cram" at this stage of your preparation. Select the most important topics (those that are most heavily covered on the exam), and let the rest go.

Do not study intently the last day or two before the exam. Your ability to answer questions correctly will be greater because your analytical ability will be high and not overridden by specific recall from the last day's cramming.

Practice the Exam Process

Set aside some time shortly before the exam to run through multiple-choice questions and answers (as many as you can). If you haven't taken this type of exam for a while, you need to retrain your mind in this type of test-taking.

In addition to answering the multiple-choice questions throughout this text, go through the current *PEER* products put out by ACEP; some of those questions have been on previous exams. Most written board courses have pretests (and post-tests) that amount to a couple of hundred or more multiple-choice questions.

Go through the questions as if you were actually taking the exam. Answer all the questions you know first. Go back later to work on the questions you didn't know or were unsure of. There is a reason for this based on test-taking psychology: answering questions you are sure of first increases your confidence in answering questions you are unsure of later. If you are still unsure of some questions when you return to them, try using the techniques described in the section "How to Take a Multiple-Choice Exam."

